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EATING ATTITUDES, BODY IMAGE PERCEPTION, PERSONALITY AND ENDOCRINE PROFILE IN MALE HABITUAL LONG DISTANCE RUNNERS

by

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A THESIS
SUBMITTED TO THE FACULTY OF GRADUATE STUDIES
AND RESEARCH IN PARTIAL FULFILLMENT OF THE
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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled EATING ATTITUDES, BODY IMAGE PERCEPTION, PERSONALITY AND ENDOCRINE PROFILE IN MALE HABITUAL LONG DISTANCE RUNNERS: An investigation into the similarities of behaviours, personality and endocrine status between male runners and Anorexia nervosa patients, submitted by GARRY DAVID WHEELER in partial fulfillment of the requirements for the degree of Master of Science in Physical Education.

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Date MARCH 12 1984

ABSTRACT

There has been a growth in opinion that habitual or obligatory long distance running bears remarkable similarities to the phenomenon of Anorexia nervosa (AN) (Sours, 1980, 1981; Yates, Leehay and Shisslak, 1983).

The purpose of the study was thus to investigate a hypothesis of behaviour, psychological and endocrine similarities between runners, non-runners and previous findings in anorexic patients.

To investigate the hypothesis eating attitudes, personality, body image and endocrine status were assessed in a group of 49 runners, and 18 non-exercising controls after a 24 hour rest from running. Runners were grouped according to weekly mileage and analysis was conducted through three stages of mileage grouping. Major findings in the study appeared through a three group analysis using a 40 plus miles per week (high mileage), 20-39 miles per week (low mileage) and control grouping method.

The high mileage runners significantly overestimated waist width compared to controls ($p < 0.05$) and scored significantly higher infrequency scores on the Jackson Personality Inventory ($p < 0.05$). No differences were found among groups on eating attitudes. Free and non-SHBG bound testosterone and prolactin levels were significantly lower in the high and low mileage runners than controls ($p < 0.05$). Cortisol, LH, FSH and thyroid hormone levels were normal at basal levels in the runners.

Correlations were computed between selected measured variables. High waist estimation scores were correlated with high anxiety and infrequency scores in the high mileage runners ($p < 0.05$). Inverse

correlations were found between waist perception and self-esteem in high mileage runners ($p < 0.05$) and between waist perceptions and total, non-SHBG bound and free testosterone in the low mileage runners ($p < 0.01$). Anxiety was inversely correlated to non-SHBG T and infrequency was inversely correlated to free T in the high mileage runners ($p < 0.01$). The correlations computed, however, did not provide a consistent picture in terms of the relationship between mileage, ponderal index, body image, eating attitudes, personality and hormone levels.

A hypothesis of anorexic behaviours and attitudes in runners as measured by the Eating Attitude Test, running commitment questionnaire and Jackson Personality Inventory was not supported. While providing evidence of possible alterations in the pituitary-gonadal axis or in the metabolic clearance and production of testosterone, the hormone profile of the runners did not resemble that of anorexics.

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CHAPTER I

INTRODUCTION

In the late 1960's and early 1970's it became clear that the general physical condition of many people was extremely poor. The increased mechanization of the late 20th century has permitted a degree of inactivity which has led to deteriorating cardio-pulmonary health and increasing obesity. This has resulted in the tremendous increase of interest in physical fitness since 1970.

Cooper's 'The New Aerobic' (1973) was a typical work supporting the new attitude towards a need for physical activity in the early 1970's. Recognizing the increasing incidence of cardiovascular and cardio-pulmonary disease he observed,

> Heart disease is a national disaster. Every year nearly a million Americans die from heart and blood vessel diseases (p. 11).

Since Cooper's work, much has been published in relation to methods of achieving fitness and a large number of health spas, fitness clinics and physical training centres have opened up in response to the call for physical fitness. A whole industry of running equipment, weight training equipment and weight loss aids has grown with this new awareness. Both the fitness movement and the wealth of popular literature on fitness are based on a firm belief that physical activity of a regular nature can improve the quality of life.

Many statements have been made regarding fitness but perhaps the most popular are that exercising prolongs your life or paradoxically that the purpose of fitness and regular activity is not to add years to life but to add life to years.

Perhaps the most popular form of exercise adopted by millions today is that of running. What was a 'jogging' fashion of the early 1970's is now a serious 'running' phenomenon, one on which this work is based. Magazines such as Runner's World, Canadian Runner, The Runner, and Running have a global circulation and the philosophy of the 'running Doctor', Dr. George Sheehan of Runner's World, 'Running and Being' (1978) is upheld by thousands who run religiously everyday.

Running, as a way of life, is seen by some as a 'positive addiction' (Glasser, 1969) although today the concept of 'Negative Addiction' (Morgan, 1979) and neurotic illness in exercising enthusiasts (Little, (1981) alludes to the disturbing degree to which some have adopted running and habitual exercise.

The new fashion regarding slimness and diet and exercise fads parallels an increase in Anorexia nervosa (AN), a disease considered rare as little as 40 years ago (Ingram, 1978) and traditionally associated with women. This self-imposed starvation disease may well be attributable to psycho-sociological factors related to the desire for thinness in young and middle-aged alike (Bruch, 1973).

Perhaps more disturbing is a detectable trend since 1980 towards linking AN and compulsive exercise (Smith, 1980; Sours, 1980, 1981; Yates, Leehay and Shisslak, 1983). The increase in both AN and physical exercise over the last decade and parallels between the two phenomena have prompted this study.

A Statement of the Problem

Observation, interviews and an extensive review of literature have revealed five important issues on which this study is based.

1. There has been an increase in long distance running during the last decade.

2. Social pressures on young men and women may be responsible for a dramatic increase in AN. For example, the diet cult and desire for 'slimness'.
3. Until comparatively recently AN has been considered primarily a female phenomenon. It has been recognized that males succumb to the disease (Bruch, 1978).
4. Doctors and psychologists are becoming aware that running may develop into an obsessive, compulsive addiction (Morgan, 1979; Little, 1981; Smith, 1980; Yates et al., 1983).
5. There are similarities between the ritualistic behaviors associated with running and AN and in social background between AN patients and runners.

From those observations two important questions have arisen.

(i) Is running an expression of the hyperactive state of male and female anorexics?

(ii) Does running that develops into an obsession, lead to the development of anorexic tendencies or AN itself? More specifically, does running lead to the development of AN in males?

The Purpose of the Study

The purpose of the study was to ascertain whether male runners exhibit a similar behavioural, psychological and endocrine profile to male and female anorexics. To investigate this, measurements were made of eating attitude, body image perception, personality and hormone levels (T_3, T_4, testosterone, free testosterone, non-SHBG bound testosterone and cortisol, LH, SFH and prolactin).

A Justification of the Study

The review of literature will deal with the theoretical background underlying the study. However, at this point certain issues will be mentioned by way of justification of the intended study.

Today's social demands for a slimmer physique in both the male and female may well be responsible for a parallel increase in the development of Anorexia nervosa and compulsive, addictive, habitual or obligatory running.

Since 1980 there has been a growth in the literature that considers running to be harmful for a number of reasons. This new perspective is not related to the anatomical damage that perpetual daily running may cause but rather to psychological dependency, even 'addiction'. Whereas running has been labelled as a form of "positive addiction" (Glasser, 1976) others have called compulsive running a "negative addiction" (Morgan, 1979). Cessations of running or habitual exercise due to injury has also been associated with neurotic illness and serious withdrawal symptoms (Little, 1981; Sours, 1981).

Of greater concern, habitual running has been linked with AN, particularly in males (Yates, Leehay, Shisslak, 1983). One study links the food aversion of the young athlete striving to lose weight to the food avoidance of the anorexic female (Smith, 1980). Another stresses the similarities of female athletes and AN patients (Henry, 1982).

Since 1980, some authors have observed remarkable similarities between the habitual or compulsive exerciser and the anorexic male and female. Similarities that are both, psychological, psychosomatic and behavioural have been observed (Norval, 1980; Sours, 1980, 1981; Yates, Leehay, Shisslak, 1983).

To date, however, the conclusions of such studies are based purely on anecdotal evidence and questionnaire responses. As yet, a thorough basal endocrine profile of male habitual runners has not been studied and thus a valid comparison cannot be made between AN and habitual running.

A Theory of Andro-Anorexia Athletica - or Anorexic Tendencies in the Male Habitual Long Distance Runner

From the foregoing discussion two possible theories have evolved.

1. (a) Habitual running is a manifestation of the hyperactive condition associated with AN. Males who have anorexic tendencies or who are anorexic, run to burn-off unwanted calories through continual activity.

   (b) Endocrine dysfunctions may be found in male runners and are analogous to those found in the male AN patient.
2. (a) A runner may develop obsessive qualities associated with running and subsequently adopt an abnormal degree of food awareness, a distorted body image and rituals simulating AN.

   (b) Endocrine dysfunctions may occur in the male runner secondary to a loss of body fat as a result of dietary manipulations. This may be manifest by the reduced LH and testosterone levels found in male anorexics as a result of an impaired hypothalamic-pituitary gonadal axis.

The Hypothesis

The author intends to adopt the latter theoretical position on which to base the foregoing study. A hypothesis may thus be stated.

Long distance running of an habitual nature may lead to a preoccupation with diet and bodily dimensions as weekly mileage increases manifest by abnormal eating attitudes and a distorted body image.

Habitual exercise also leads to the development of endocrine disorders in the male runner equivalent to those found in male and female AN patients.

Statistical Hypothesis

(a) $H_0 : M_1 = M_2 = M_3 = M_4 = M_5$

$H_1 : M_1 \neq M_2 \neq M_3 \neq M_4 \neq M_5$

M_1 = university runners (comparative)

M_2 = 70 miles plus/week club runners

M_3 = 40-69 miles/week club runners

M_4 = 20-39 miles/week club runners

M_5 = non-exercising controls

(b) $H_0 : M_1 = M_2 = M_3 = M_4$

$H_1 : M_1 \neq M_2 \neq M_3 \neq M_4$

University runners are regrouped according to mileage into one of other three categories.

(c) $H_0 : M_1 = M_2 = M_3$

$H_1 : M_1 \neq M_2 \neq M_3$

M_1 = 40 miles plus/week runners

M_2 = 20-39 miles/week

M_3 = non-exercising controls

A Definition of Terms

1. Anorexia Nervosa (AN)

AN is a disorder primarily affecting young women, although the incidence in the male is increasing. It is characterized by marked self-induced weight loss, a distorted body image, an aversion to food and a relentless pursuit of thinness. AN patients are often hyperactive and will deny the existence of any illness even in the face of severe emaciation (Bruch, 1978).

The onset of the disorder usually occurs during the teenage period at adolescence although it may manifest itself in persons as old as 40 years of age. The prevalence in white women around puberty in Western countries may be as high as one in two hundred (Schwabe, 1981) with a ratio of between one in ten and one in twenty males to females (Bruch, 1973). Anorexia nervosa has been described as a 'relentless pursuit of thinness' (Bruch, 1983), 'Almost invariably a state of weight phobia' (Crisp, 1970) and 'A morbid fear of being fat' (Russell, 1970).

2. Bulimia Nervosa

Bulimia is a condition in which young women in particular engage in gorge-vomit behaviour. Like AN, the patient has a desire for thinness but unable to control the need for oral gratification, caloric absorption is arrested via self-induced vomiting.

3. Hormones

Hormones are substances secreted by endocrine glands (hypothalamus, pituitary, thyroid, testes, ovaries and adrenal cortex and medulla). They are released into the extracellular spaces surrounding the gland cells and are absorbed into the blood and carried to all parts of the body. Each specific hormone has particular target tissues on which it acts and has a particular effect.

(a) Testosterone

This is a steroid hormone secreted mostly by the interstitial cells of the testes (95%) and to a lesser extent by the adrenal glands (5%). It functions to produce the secondary sex characteristics of the male via its androgenic action and may be related to potency. The anabolic action of the hormone is important in stimulating protein synthesis through the activation of cyclic AMP.

(b) Protein Binding of Testosterone

Testosterone circulates in blood partially bound to proteins with only a small fraction free. The largest portion (approximately 60%) is bound to sex hormone binding globulin (SHBG), with the majority of the remainder attached to albumin. The SHBG has a high affinity, low volume binding to testosterone while albumin has a low affinity, high volume relationship. Protein binding modifies the biological availability of testosterone so that SHBG bound testosterone is not bioavailable. Free testosterone is available to cells and it remains open to discussion whether albumin bound testosterone is available.

(c) Follicle Stimulating Hormone (FSH) and Luteinising Hormone (LH)

Both hormones are regulators of gonadal function and are therefore gonadotropins. LH regulates androgen production from the interstitial cells. The androgen is used peripherally as described above and with FSH is involved in the production and maturation of sperms with the semeniferous tubules. In women LH also regulates androgen production as precursors for estrogen production and for secretion into the general circulation. LH is also required for ovulation and the conversion of the ovulatory follicle into the corpus luteum.

(d) Cortisol

Cortisol is a glucocorticoid secreted by the adrenal cortex and also one of the steroid hormone group. Secretion of cortisol is controlled almost entirely by adrenocorticotropic hormone (ACTH) secreted by the anterior pituitary gland. ACTH, also called corticotropin, also enhances the production of adrenal androgens. ACTH secretion is controlled by corticotropin releasing hormone (CRH). It is secreted into the primary plexus of the hypophysical portal system in the median eminence of the hypothalamus and then carried to the anterior pituitary gland where it induces ACTH secretion. Cortisol is an important factor in carbohydrate and fat metabolism as well as being a stress related hormone and anti-inflammatory agent.

Effects of Cortisol on Carbohydrate Metabolism

Cortisol (and other glucocorticoids) stimulate gluconeogenesis by the liver as much as six to tenfold. Cortisol increases the transport of amino acids from the extracellular fluids into the liver cells, increasing the availability of amino acids to cells for conversion to glucose. Enzymes required to convert amino acids into glucose are increased in the liver cells, an effect mediated by cortisol. Cortisol also mobilizes amino acids from the extrahepatic tissues and as a result more amino acids are available in plasma to enter into the gluconeogenesis process of the liver. Increased gluconeogenesis increases the glucogen in the liver cells.

Cortisol also causes a moderate decrease in the rate of glucose utilization by the cells. The cause for this decrease is unknown but it may be that glucocorticoids depress the oxidation of NADH. NADH must be oxidized to allow glycolysis.

Glucocorticoids are also known to slightly depress glucose transport into the cells.

Effects of Cortisol on Protein and Fat Metabolism

A principle effect of cortisol on the metabolic systems is reduction of protein stores in most body cells except the liver. This is caused by decreased protein synthesis and increased catabolism of protein already in the cells. Liver proteins are enhanced and plasma proteins produced by the liver are enhanced. This effect is probably caused both by the ability of cortisol to enhance amino acid transport into liver cells and by enhancement of the liver enzymes required for protein synthesis. While transport of amino acids into extrahepatic cells is reduced, catabolism of proteins in the cells continues to release amino acids from the already existing proteins, and these diffuse out of the cells to increase the plasma amino acid concentration. Cortisol is thus said to mobilize amino acids from the tissues.

Cortisol also acts to promote mobilization of fatty acids (FFA) from adipose tissue but only to a small extent. This increase in FFA in the plasma does, however, increase their utilization for energy. Cortisol also moderately enhances the oxidation of fatty acids in the cells as well. The fat mobilizing effect of cortisol is an important factor for long-term conservation of body glucose and glycogen.

Persons with excess cortisol secretion frequently develop a peculiar type of obesity with excess deposition of fat in the chest and head regions of the body. This obesity appears to result from excess stimulation of food intake so that fat is generated in tissue faster than it can be mobilized in the body.

Function of Cortisol in Stress

Almost any type of stress, whether physical or neurogenic will cause an immediate and marked rise in ACTH. This is followed by a greatly increased adrenocortical secretion of cortisol. The value of a raised cortisol level in times of stress is unclear. However, raised cortisol levels are associated with increased protein and fat mobilization and utilization and therefore of benefit to the body in terms of energy supply.

Antiinflammatory Effects of Cortisol

When the body experiences trauma such as tissue damage, cortisol is released which has an effect of blocking early stages of the inflammation process. If inflammation has already begun, it causes rapid resolution of the inflammation and increased rapidity of healing.

(e) Thyroxine (T_4), Tridothyronine (T_3), Thyroid Stimulating Hormone (TSH) and Reverse T_3 (rT_3)

T_4 is secreted from the thyroid gland in response to thyroid stimulating hormone from the anterior pituitary gland. T_3 is mostly produced by peripheral de-iodination of the T_4. TSH controls the secretion of hormones from the thyroid gland by causing the amount of cyclic AMP within the thyroid cells to increase. TSH secretion itself is controlled by thyrotropin releasing factor (TRF) released from the hypothalamus. As blood concentrations of thyroid hormones (T_3, T_4) increase the secretion of TSH and TRF are reduced by a negative feedback mechanism. T_3 and T_4 act to increase the rate at which energy is released from carbohydrates and the rate at which proteins are synthesized in body cells. Peripheral T_4 to T_3 conversion is reduced with caloric and protein malnutrition when T_4 is converted to the physiologically inactive stereoisomer reverse T_3 (rT_3).

4. Body Image

Body image is defined by Schilder (1935) as,

> The picture of our own body which we form in our minds. That is to say, the way in which the body appears to ourselves.

The body image may be thought of as the individual's perception of his or her body in relation to the individual concept of ideal body form.

Body image was measured by an Image Drawing projection method (Askevold, 1975) and was expressed as a Body Image Perception Index (BPI) as defined by the following formula:

$$BPI = \frac{\text{Perceived Size}}{\text{Real Size}} \times \frac{100}{1}$$

Scores of over one hundred were indicated as an overestimation of body size and less than one hundred, as underestimation of body size. The test comprised measures of shoulder, waist and hip width and provided a composite mean for all scores.

5. Eating Attitudes

Eating attitudes were evaluated by the Eating Attitude Test (Garner and Garfinkel, 1979). Abnormal eating attitudes were defined as scores of 30 plus on the test.

6. Habitual Running

Habitual running was defined according to a mileage of at least 20 miles per week, accumulated over 5 or more runs.

7. Personality

Personality was measured by the Jackson Personality Inventory. Personality was described in terms of 16 sub-scales.

8. Negative Addiction

Negative addiction is the process by which running becomes a dominant, all encompassing influence in the life of the runner. Internal feelings of well being and self-righteousness dominate external issues such as the marriage, family and occupation. The addiction process may be considered complete when a runner continues to run even against medical advice (Morgan, 1979).

Limitations and Delimitations of the Study

Delimitations

1. Male runners covered between 20 and 70 plus miles per week.

2. Runners were divided into 4 groups according to the following criteria:
    - (a) University (competitive runners),
    - (b) 70 plus miles per week,
    - (c) 40-69 miles per week, and
    - (d) 20-39 miles per week.
3. Each group was comprised of 20 runners.
4. A control group of 20 non-exercising males were selected. The criterion for selection was lack of interest and of participation in physical activities.
5. The study was ex-post facto in nature and was designed to examine group differences in body image, eating attitudes and hormone levels and to ascertain the degree to which the dependent variables were related (correlated).

Limitations

1. The study was ex-post facto (correlational) and descriptive, therefore a cause/effect relationship could not be established beyond a tentative degree.
2. Subjects 'self selected' into groups. There was no random assignment of subjects to group or treatment.
3. Dependent variables were measured after the fact.
4. The study was limited to one sex and may therefore only be generalized to males.
5. The possibility of attrition between test booking date and the test date. This decreased the reliablity and validity of test measures and correlations.
6. The base rate of Anorexia nervosa in the population was unknown.

7. The study rested on the assumption of habitual running being partly due to a general desire to "get in shape" or more specifically, to lose weight.
8. The study relied in part on information obtained by questionnaires which were limited in their value to research in the following ways:
   - (i) Misinterpretation of questions,
   - (ii) Failure of subjects to answer true/false questions with integrity,
   - (iii) The fatigue effect of long questionnaires, and
   - (iv) The denial aspect of the illness, Anorexia nervosa.

     If runners did have true AN tendencies they may well have adopted a defensive attitude in questions relating to self-esteem, confidence, anxiety and eating attitudes.

The Dependent and Independent Variables

Independent Variables

Running and mileage per week constituted the independent variable. This variable was divided into 4 groups, namely:

X_1 = university/competitive runners
X_2 = 70 miles/week plus runners
X_3 = 40-69 miles/week
X_4 = 20-39 miles/week

The variables X_1, X_2 and X_3 were amalgamated via two stages during analysis to ascertain the degree to which mileage was a significant factor in body image, personality, eating attitudes and hormone levels.

Dependent Variables

The dependent variables were:

(a) Eating Attitude Scores,

(b) Body Image Perception Index,

(c) The Personality Profile, and

(d) Hormone Levels at Rest.

The above variables are described in the methodology section.

CHAPTER II

A REVIEW OF LITERATURE

PART I

Anorexia Nervosa: History, Symptomology and Endocrine Dysfunctions In Male and Female Anorexia Nervosa

Anorexia nervosa (AN) is a complex psychosomatic disorder with secondary physiological and endocrine complications. It is only recently, however, that clear distinctions have been made between primary hypothalamic disorders and Anorexia nervosa and its attendant secondary endocrine disorders. In the following discussion the author will, by way of introduction to the concept of Anorexia nervosa tendencies in habitual runners, discuss theories of the genesis of the AN disorder.

Anorexia nervosa is primarily a disorder affecting young women, although its occurrence in the male population is increasing (Bruch, 1973; Hogan, Huerta and Lucas, 1974). It is characterized by a variety of psychological abnormalities, such as a distorted body image, bizarre eating attitudes and behaviours, a denial of illness and secondary physiological dysfunctions manifest by severe cachexia and endocrine disorders. It has been defined as "a relentless pursuit of thinness" (Bruch, 1973) and "a condition where the subject starves herself literally to death" (Bruch, 1978). Crisp (1970) calls it, "almost invariably a state of weight phobia", and Russell (1970), "a morbid fear of being fat". The prognosis for the patient is poor and patients may be cured temporarily only to revert back to anorexic behaviour (Dally, 1969) and some may even die. Dally (1969) considers a 10-15% mortality rate as realistic although it may be as high as 50%. Warren and Vand-Wiele

(1973) report the death of 3 subjects out of 42 in their study (7.1%).

A brief examination of the historical development of theories relating to the causes of AN shows that the literature has come full circle, that is to say early psychological theories, later replaced by organic theories of origin, have now once again gained acceptance.

The disease was first described by Richard Morton (1689) who termed it a nervous consumption. Later, William Gull (1874) coined the phrase "Anorexia nervosa" at about the same time as Lasègue (Paris, 1873) coined the term "Anorexia Hysterique", to describe the hysterical aversion to food. The early recognition of a primarily psychological disorder was overshadowed by Morris Simmonds (1914) who attributes the disorder to a hypothalamic-pituitary lesion. The notion of organic, rather than psychological origin, persisted until around the late 1930's when the work of those such as Farquaharson and Hyland (1938), Richardson (1939) and McCullagh and Tupper (1940) revived the theory of a primary psychological disorder. The work of the above-mentioned authors made a distinction between true primary AN and hypothalamic-pituitary (organic) disorders in that AN was associated with severe weight loss through food avoidance with a paradoxically high state of hyperactivity while hypothalamic-pituitary disorders with a structural pathology were accompanied by a less severe weight loss and a high degree of lassitude.

Since 1960 much work has been done to differentiate between 'True' or 'Primary' Anorexia nervosa associated with a relentless pursuit of thinness, food aversion and a distorted body image from other disorders resulting in secondary or atypical AN, caused by organic and other psychological disorders such as Schizophrenia and chronic depression, of a temporary nature (Bruch, 1973).

A New Psycho-Sociological Approach

Anorexia nervosa was rarely diagnosed forty years ago, yet today the apparent incidence of the disease is increasing (Schwabe, 1981). The prevalence may be as high as one per two hundred women with a cultural and psychological basis (Shainess, 1979).

On the increasing incidence of the disease, Bruch (1978), observes,

> One might speak of an epidemic illness only there is no contagious agent, the spread must be attributed to psychosocial factors (p. 8).

Society no longer values obesity as a sign of opulence; slimness is a new value in society. Contemporary cultural attitudes provide strict limitations of acceptable body fatness in young women (Ingram, 1968) and Bruch (1978) attributes the disease, in part to the tremendous emphasis that society places on slimness. She also observes that the media, particularly television, insists persistently that "one can be loved and respected only when slender" (p. 8).

Associated with the new cultural awareness of slimness is an ever-growing diet industry with as many dietary fads. It has been observed that claims associated with products designed to induce weight loss, range from serious well-documented scientific studies to unscrupulous publicity stunts (Bruch, 1973, p. 309).

Anorexia Nervosa In the Male

Anorexia nervosa is less common in the male than in the female. Bruch (1973) estimates the male/female ratio as between one in ten to one in twenty and Ushakov (1970) considers one in five to be a more realistic figure. This latter view may be correct since the medical profession has been reluctant to make the diagnosis in males (Hay and Leonard, 1979; Bruch, 1973; Hogan, Huerta and Lucas, 1973).

Cessation of menstruation or failure of the onset of menses is an immediately recognizable disorder which aids the diagnosis of AN in young girls and post pubertal women. This factor has been included as one of the diagnostic criteria of AN and has thus hampered diagnosis in males by virtue of eliminating them from the disorder by definition (Dally, 1969). Thus, Cobb (1943), Nemiah (1950), Selvini (1965), Kidd and Wood (1966) and Palozzi (1971) deny the existence of the disorder in the male. Palozzi (1971) calls AN-like tendencies in the male, "Pseudo-Anorexia nervosa".

However, it is now thought that the disease is more common in males than was previously recognized (Hay and Leonard, 1979; Crisp and Toms, 1972). Bruch (1971) observes that the major diagnostic problem in relation to AN in males is the same as for females, in that 'True' or 'Primary' AN is often confused with what she terms 'Secondary' or 'Atypical' AN.

A Comparison of the Symptoms of AN in Males and Females

The symptoms of the disorder are essentially the same in the male and female (Bruch, 1971). That is to say, a pursuit of thinness, food avoidance, distorted body image, denial of illness and accompanied frequently by a hyperkinetic state (Hay and Leonard, 1979; Beumont et al., 1972). The major difference is that of the absence of amenorrhea in the male. Some of the signs and symptoms will be examined in the following discussion.

Both male and female anorexics engage in complex ritualistic behaviours limiting caloric intake. Both male and female AN patients develop an aversion to the intake of food (Bruch, 1971).

The hyperactive state of the patient with AN has been described as "goalless activity to burn off calories" (Bruch, 1971, p. 44). AN patients often engage in a ritual of compulsive exercise in order to aid weight control (Hogan, Huerta and Lucas, 1972) although as Bruch (1971) observes this is often ignored as a diagnostic criterion.

A distorted body image is an important indicant of the psychosomatic imbalance of the anorexic patient. Schilder (1935) provides a definition of body image.

> . . . the picture of our own body which we form in our mind, that is to say, the way in which the body appears to ourselves (p. 1).

The AN patient typically overestimates the width of his or her body when in fact they are in a severely emaciated condition. This has been interpreted as a manifestation of the anorexic's attitude towards an inner desire for thinness (Slade and Russell, 1973; Askevold, 1977; Button, Fransella and Slade, 1977; Pierloot and Houben, 1978; Garner et al., 1978). Many methods have been employed to assess the degree to which the body image is distorted and these may be summarized by the following:

METHOD	REFERENCE
Draw-A-Person Test	Machover (1947)
Body-Image Boundary Score	Fisher and Cleveland (1958)
Questionnaire-Satisfaction with Body Assessment	Fisher (1964)
Adjusting Body Distorting Photograph	Taub and Orbach (1964)
Tactile Size-Estimation Task	Shontz (1969)
Visual Size-Estimation Task	Reitman and Cleveland (1964) Slade and Russell (1973)
Tactile Size Estimation and Projection (Profile Drawing)	Askevold (1975)

The purpose of these methods is to assess an individual's perception of his or her body. Results are more commonly expressed as a Body Perception Index (BPI) (Reitman and Cleveland, 1964; Slade and Russell, 1973) through the following formula:

$$\text{BPI} = \frac{\text{Perceived Size}}{\text{Real Size}} \times \frac{100}{1}$$

A score of one hundred plus is indicative of overestimation and less than one hundred, underestimation.

The degree to which AN patients overestimate their size varies and this may reflect the severity or duration of the illness (Button, Fransella and Slade, 1977). Slade and Russell (1973) found that anorexics overestimated their body dimensions by between 28-59%, Garner et al., (1976) 12-27% and Casper et al., (1979) 19-27%. However, overestimation of body size may not be specific to AN (Pierloot and Houben, 1978). Button, Fransella and Slade (1977), for example, found that although AN patients in their study overestimated by between 2 and 23%, normal individuals overestimated by 7-31%. However, the overestimation of body size may be considered to be far more serious in those in an emaciated condition than for normal individuals. Discrepencies in the results of studies probably arise from the variety of methods used in estimation of body dimensions. Most researchers have used visual perception type methods which do not involve actual projection of the body image. The need for bodily involvement in projecting the body image is assessed by Witkin (1965), who observes that direct bodily involvement in an assessment of body image requires few inferences than are necessary when the body image is assessed from estimation-type methods and conversation or questionnaires about the body.

Little, however, has been done with regard to body image assessment in the male anorexic. This situation exists for the same reason that few males are diagnosed as anorexic; that is to say, that until very recently the medical profession has been reluctant to acknowledge true male AN.

Endocrine Dysfunctions Associated with Anorexia Nervosa

To this point, the foregoing discussion has dealt with the psychological aspects of AN. However, endocrine disorders are important for the diagnosis of the disease, particularly in relation to amenorrhea in women. Amenorrhea and the endocrine mechanisms associated to this dysfunction may thus be examined.

Secondary amenorrhea is a common feature of AN (Garfinkel and Garner, 1982), while primary amenorrhea is much less common, occurring in only 4% of AN patients (Garfinkel, Moldofsky and Garner, 1980).

The pathophysiological basis of amenorrhea is unclear since both weight and emotional stress may be involved. According to Fries (1977) and Hurd et al. (1977) the onset of secondary amenorrhea occurs in 70% of cases after weight loss has occurred while Fries (1977) observed amenorrhea in 24% of cases prior to weight loss. In contrast to this, Bass (1947) noted that emotional stress was an important contributory factor since 60% of women in a concentration camp ceased menstruation prior to starvation. Garner and Garfinkel (1982) reviewed the literature and concluded that weight loss was the most important factor. Loss of body fat is related to overall weight loss, and the concept of a critical percentage of body fat being necessary for the onset and maintenance of the menses has been considered (Frisch, 1977).

It is unclear exactly what causes amenorrhea, although it is most likely a combination of weight loss, decreased body fat, caloric imbalance, dietary deficits and emotional stress. Garfinkel and Garner (1982) thus conclude that a minimum fat level is a necessary but insufficient condition for the onset and maintenance of normal menses.

LH and FSH Levels

LH and FSH levels are reduced in AN patients (Boyar, 1974, 1978; Kalucy and Crisp, 1976; Brown et al., 1977). It has also bee noted that LH levels are more markedly reduced than FSH levels (Warren and Vand Wiele, 1973). The 24 hour secretory patterns of LH and FSH have been found to resemble those found in pre- to mid-pubertal girls (Katz et al., 1976). Both LH and FSH levels return to normal on refeeding (Boyar and Katz et al., 1974). Hypopituitary function is not considered as primary to AN in that LH and FSH responses to Luteinizing Hormone Releasing Hormone (LHRH) in provocative tests, although reduced, do occur (Nillius and Wide, 1972; Mecklenberg et al., 1974; Garfinkel and Garner, 1982). This reflects inhibition of Gonadotrophic releasing hormone release at the hypothalamus and results in cessation of cyclic ovarian function. Warren et al. (1973) thus postulate the presence of a primary hypothalamic lesion in AN.

Growth Hormone

Resting levels of growth hormone (GH) are elevated in AN patients (Brown et al., 1977; Garfinkel et al., 1975) and it is suggested that the elevated GH levels are related to decreased caloric intake (Garfinkel et al., 1975). Diseases related to caloric and protein starvation are typically associated with raised GH levels (Pimstone, Becker and Kernoff, 1973).

Pituitary-Thyroid Function

Thyroxine (T_4) levels are in the normal range (Brown et al., 1977; Boyar et al., 1977) or at the lower end of the normal range (Hurd et al., 1977). Others have found lower than normal values (Kingstone and Boss, 1979). Serum Triodothyronine (T_3) levels have generally been found to be reduced in AN (Mosthang et al., 1975; Hurd et al., 1977; Miyai, 1975) and this decrease has been associated with a corresponding increase in the inactive stereoisomer, reverse T_3 (rT_3) (Burman et al., 1977). High rT_3 levels in AN are similar to findings in people suffering from wasting diseases associated with caloric deprivation (Vagenakis, 1977; Chopra and Smith, 1975; Boyar et al., 1977) and carbohydrate restriction (Spaulding et al., 1976). Such changes are normalized by refeeding and subsequent weight gain (Isaacs et al., 1978; Wakeling et al., 1979).

Basal thyroid stimulating hormone (TSH) levels appear to be normal in patients suffering from AN (Beumont et al., 1976; Brown et al., 1977) although both increased (Aro, Lamberg and Pelkonen, 1975) and decreased (Hurd et al., 1977) levels have been reported. Since TSH levels are normal it is unlikely that thyroid changes are due to Primary Thyroid failure (Garfinkel and Garner, 1982). TSH responses to thyrotropin releasing hromone (TRH) are normal although delayed (Beumont et al., 1976). TSH responses to TRH return to normal on refeeding (Vigersky, 1977; Leslie et al., 1978).

In conclusion, Garfinkel and Garner (1982) note that pituitary function is intact in AN and that changes in the hypothalamic-pituitary-thyroid axis are likely to occur as an adaptation to chronic illness and starvation.

Pituitary (Adrenocorticotrophic Hormone)-Adrenal Function

Elevated levels of plasma cortisol are found in AN (Alvarez, 1972; Brown et al., 1977) and the diurnal variations of cortisol are flattened (Garfinkel et al., 1975). Others have reported normal levels in AN patients (Vigersky, 1977).

The mechanism of elevated cortisol levels is related to T_3 deficiency. Reduced T_3 levels result in elevated cortisol levels because of decreased clearance (Boyar et al., 1977). Administration of T_3 results in increased cortisol clearance and levels return to normal.

Prolactin Levels

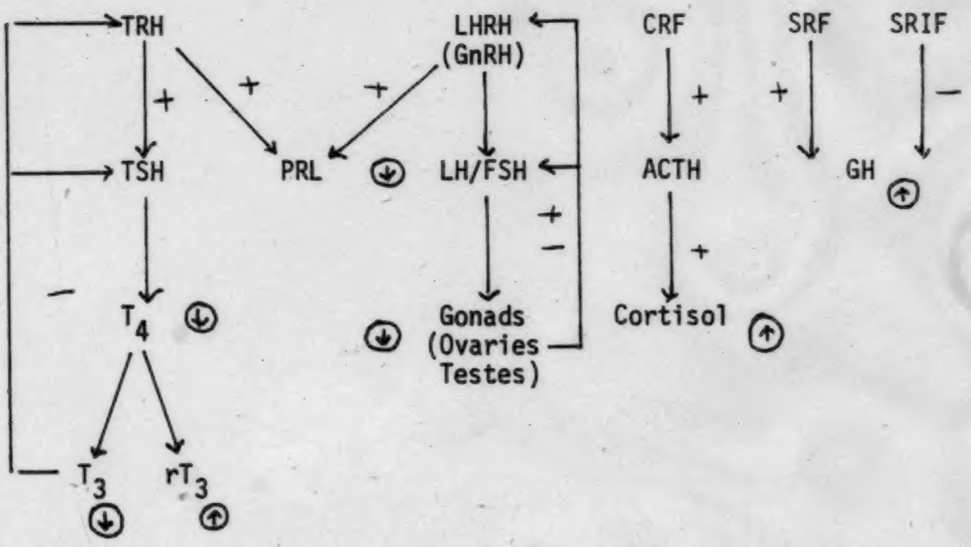
Prolactin levels and response to provocative testing are both normal in AN (Beumont et al., 1974; Isaacs et al., 1980; Garfinkel and Garner, 1982). The latter authors conclude that increased prolactin is not responsible for secondary amenorrhea in AN patients.

Testosterone Levels

Reduced urinary testosterone (T) levels have been found in male AN patients (Beumont, 1970, 1972; Davidson, 1976; Frankel and Jenkins, 1975; Garfinkel et al., 1975; Ismail and Harkness, 1967) while serum and urinary output in females are normal (Casper et al., 1979) or elevated (Baranowska and Zgliczyniski, 1979). Abnormalities in T levels are normalized on refeeding (Buvat et al., 1983; Beumont et al., 1970, 1972).

In summary, endocrine dysfunctions in AN appear to relate to weight loss and disturbances in the hypothalamic pituitary gonadal, pituitary adrenal, pituitary thyroid axes and gonadotrophic hormone release.

Summary of Hormone Feedback Systems


TRH
TSH
T_4
T_3
rT_3
PRL
LHRH
(GnRH)
LH/FSH
Gonads
(Ovaries
Testes)
CRF
ACTH
Cortisol
SRF
SRIF
GH
+
−

Legend:

TRH	Thyroid releasing hormone
TSH	Thyroid stimulating hormone
T_4	Thyroxin
T_3	Triodothyronine
rT_3	Reverse Triodothyronine
LHRH (GnRH)	Luteinising hormone - (gonadotrophin releasing hormone) releasing hormone
LH	Luteinising hormone
FSH	Follicle stimulating hormone
CRF	Corticotropin releasing factor
ACTH	Adrenocorticotropic hormone
Cortisol	
SRF	Somatotropin releasing factor
SRIF	Somatostatin releasing inhibiting factor
GH	Growth hormone

Positive/Negative Effect	+ —
Effect Direction	↓
Increased/Reduced Levels	(↓)

Garfinkel and Garner (1982) conclude that there is considerable evidence to suggest the presence of hypothalamic disturbances in patients with AN. The endocrine dysfunctions occur through a variety of mechanisms and are generally reversible.

Endocrine Studies and Male Anorexia Nervosa

Relatively little research has been carried out on endocrine disorders in male patients, that which has been done, concentrating primarily on the gonadotrophin and adrenal hormones, that is to say testosterone, LH, FSH and cortisol. Males do not exhibit ultradian variations in gonadotrophin levels although circhoral patterns do occur. The lack of a menstrual cycle makes it impossible to use this as a diagnostic criterion (Beumont, Beardwood and Russell, 1972). An equivalent dysfunction in the male may be impaired testicular function and testosterone production. Beumont (1972) observes that the lack of potency and sexual interest in AN males might be the equivalent in the male to amenorrhea in the female.

Urinary output of testosterone per 24 hours appears to be abnormal in anorexic males. Ismail and Harkness (1966) found values of 6.2 to 31.2 μg/24 hours in anorexic patients compared to 60-130 μg/24 hours in normal patients. Davidson (1976), who reported low levels of FSH, LH and a low 24 hour urinary testosterone output in a case report of a serviceman with AN, and two studies by Beumont (1970, 1972) in 6 anorexic males, found values of 4.1 to 11.2 μg/24 hours and a mean of 8.0 μg/24 hours respectively. Both studies demonstrated that LH and FSH levels were low in male anorexics. Other studies have examined serum testosterone levels. Frankel and Jenkins (1975) found serum levels of 90 ng/dl compared to a normal range of 300-900 ng/dl and Garfinkel et al. (1975).

found levels of 76 ng/dl in their study. GH and cortisol were preported to be above normal levels in both studies.

This situation is a contrast to the raised plasma testosterone levels found in females (Baranowska and Zgliczyniski, 1979) but is consistent with reduced testosterone levels found in starvation (Gillman and Gillman, 1951; Zubiran and Gomez-Mont, 1952).

Refeeding of AN patients raises testosterone levels but not to normal levels (Beumont et al., 1970, 1972). Garfinkel et al. (1975) found an increase from 76 ng/dl to 100 ng/dl and Frankel and Jenkins (1975) found an increase from 90 ng/dl to 470 ng/dl after refeeding.

The mechanisms of endocrine dysfunctions in the male are unclear. However, the trend towards normal testosterone LH and FSH levels on refeeding suggests that the reduced weight is an important factor while the failure of hormone levels to normalize completely might indicate a primary hypothalamic disorder. Frankel and Jenkins (1975) and Garfinkel et al. (1975) consider the possibility of a hypothalamic dysfunction secondary to the problem of caloric deprivation and weight loss. Low LH levels are most likely the cause of impaired testosterone production in the anorexic male. It would seem therefore that weight loss and/or percentage body fat related to hypothalamic-pituitary, pituitary-thyroid, pituitary-gonadal axial malfunctions are important in both male and female AN. The illness in the male closely resembles that in the female, although the exact mechanism by which weight loss and endocrine disorders are linked are unclear.

PART 2

The Growth of the Running Phenomenon

Since 1970 there has been a growth of public awareness in relation to health, disease and physical fitness. The increase in cardiovascular disease as a result of sedentary occupations and excessive caloric intake has resulted in a rapidly growing diet and exercise industry.

One of the avenues of exercise chosen by millions of people world-wide is running. The benefits of running may be examined.

The Benefits of Running

The benefits of running, both physiological and psychological, are well documented. A running program results in improvements in the cardiovascular system, improvement in muscle tone, a lowering of resting heart rate, a reduction in blood pressure and increased $\dot{V}O_2$max (Matthews and Fox, 1976; Fixx, 1977). Running aids weight control by increasing the basal metabolic rate (Matthews and Fox, 1976) and may be adopted as a method of stress management and as a treatment for depression (Blue, 1979; Griest, 1979; Griest et al., 1978).

Much research has been done with regard to the psychological benefits of running. Blumenthal et al. (1982), using a 10 week jogging program with a group of non-runners versus runners, found that runners significantly lowered fatigue and tension scores on the Profile of Moods States Inventory (POMS) and scored significantly lower anxiety scores on the State Trait Anxiety Inventory (STAI). Ismail and Young (1973) reported improved emotional stability in initially unfit men after 4 months of physical conditioning and Folkins and Lynch (1972) found significantly lower depression and anxiety scores, as measured by the Multiple

Adjective Check List, in a group of school children after a sports program (n = 75) compared to a group enrolled in archery and golf courses (n = 62). Morgan and Roberts (1970) however, compared an adult exercise group engaged in various exercise programs (n = 85) to a non-exercise group (n = 16) and found no significant difference in depression scores as measured by the Zung Self-Rating Depression Scale (1965) after a 6 week training period. The authors concluded that self-report tests for normals were not sensitive to detect changes following exercise. They failed to acknowledge the possibility of there being no changes to measure.

Other studies such as that carried out by Lion (1978) on psychiatric patients showed significant improvements in psychological well being, but used too few subjects for statistical soundness, while Jorgenson and Jorgenson (1979) concluded that running was psychologically beneficial on the strength of a poor return of questionnaires.

The popular press continually extols the psychological benefits of running (Fixx, 1977; Cooper, 1968; Glasser, 1976) but Singer (1975) in an introduction to Myths and Truths in Sports Psychology aptly notes that,

> Never have so many said so much
> with so little research support.

Personality Studies of Runners, Habitual Runners and Marathon Runners

Numerous studies have been directed at defining personality profiles of long distance runners. Hartung and Farge (1977) in a study of 47 runners between 40 and 60 years old, found them to be significantly more intelligent, imaginative, reserved, self-sufficient, sober, shy and forthright than the general population, although no controls were used in the study. Jette (1975) found habitual exercisers to be more serious and tough minded than non-exercisers and Zloty and Burdick (1973) found long distance runners to be significantly brighter, imaginative and forthright than the norm, again however, failing to use controls. Other studies have utilized a pre- and post-test situation to control for the weaknesses of retrospective studies. Ismail and Trachtman (1975), Young and Ismail (1976), Sharp and Reilly (1975), Buccola and Stone (1975) and Ismail and Young (1978) found significant personality differences in relation to pre- and post-test anxiety, emotional stability and extroversion between runners and non-runners.

Personality Studies on Marathon Runners

Many devoted habitual runners eventually turn to marathon running as a competitive outlet for their training. Studies on the personality of the marathoner are few and those who have studied this group consider them to be a unique group with a unique psychological profile (Morgan and Costill, 1972).

Morgan (1968, 1974) found marathon and long distance runners to be significantly more introverted than other athletes. However, this observation was not supported by Morgan and Costill (1972) who, in a study of nine marathon runners found no significant differences in introversion and extroversion from the normal population as indicated by the Eysenck Personality Inventory (EPI) and Parallel Anxiety battery. However, the marathon runners were significantly more introverted than other sports populations. In a study of 28 elite marathoners and distance runners Morgan and Pollock (1978) found very little deviation from normal scores on a variety of personality tests including the EPI. A hypothesis of high introversion in marathon runners was not supported.

A major problem in assessing introversion and extroversion is that such measures are assumed to be indicants, purely of being shy or outgoing, sociable or lacking in social skills. Clitsome and Kostrubala (1978) used the Myers-Briggs Type Indicator (MBTI) (Myers, 1962) on 50 sub 3 hour marathoners to assess introversion and extroversion from a four-sub-class perspective, namely introversion versus extroversion, sensing versus intuitive, thinking versus feeling and judging versus perceiving. Frequencies of scores were compared using the Chi-Square statistic and significance was found between extroversion and introversion ($X^2 = 6.48$, $p < 0.01$). Thirty-four of the marathoners had scores indicating introversion ($\overline{X} = 20.24 \pm 13.32$) while 16 had scores indicating extroversion ($\overline{X} = 15.00 \pm 14.53$). A similar study by Clitsome and Kostrubala (1978) of 100 marathoners identified 54 with an extroversion preference and 46 with an introversion preference. The Chi-Square showed no significant difference between introversion and extroversion.

Demographic Characteristics of Long Distance Runners and Marathoners

It has been found that long distance runners and marathon runners are more intelligent than the population mean, better educated than the normal population and the majority occupy professional and white collar jobs (Gontang, Clitsome and Kostrubala, 1978; Clitsome and Kostrubala, 1978; Hartung and Farge, 1977). However, it should be said that runners may well be a self-selected population in that white collar professionals working in relatively sedentary occupations are more likely to run after work than the less intelligent manual labourer, who works in a physically demanding job. Those in professional occupations are also more likely to be able to afford expensive running apparel and also to engage in other 'fashionable' sports occupations. Nevertheless, the similarity between running and AN that is revealed here is that AN families are typically high achievement oriented families with the parents often working in professional occupations (Bruch, 1973, 1978). However, this too might be a self-selected population in that wealthy families would not have problems of food supply. Children from very poor families would be less likely, it would seem, to deny food in the absence of sufficiency compared to those from wealthy families who starve in the presence of plenty.

Positive Versus Negative Addiction in Runners

"Neurotic Illness in Fitness Fanatics" is the title of an article written by Little (1981), linking habitual exercise to subsequent psychological illness. The study took 3 years to complete and on the basis of his observations of fitness enthusiasts, Little concludes that any injury or illness preventing the maintenance of a regular exercise program could result in a severe mental crisis for the exercise addict. He further concludes that the contemporary craze for jogging raises serious questions about the mental health implications of this phenomenon.

To authors such as Glasser (1976) habitual running is seen as a "Positive Addiction", the psychological well-being effect outweighing serious injurious factors. Morgan (1979), however, observes that addiction to running may well be negative. Morgan considers the negative addiction process to occur in four stages. Initially, running is painful and there is a low enjoyment factor. In the second stage, as conditioning improves and running becomes less painful the enjoyment factor increases. The third stage involves increasing mileage to retain the euphoria of running accompanied by the dominance of running over external issues such as the family and vocation. The fulfillment of internal needs becomes more important than the above mentioned issue. The final stage is considered as being reached when the runner continues to run even against medical advice. Running is likened to a wonder drug, but, as a drug addict may overdose, then so the runner may overdose on running. Morgan sums up his observations by saying that the exercise addict cannot live without daily running, experiences withdrawal symptoms if deprived of exercise and continues to run even against medical advice. He concludes that, "Running is a form of negative addiction in the case of the hard core exercise addict" (p. 58).

Morgan and Little are not alone in considering the dangers of addictive exercise. Bittker (1977) observes that the adoption of a regular exercise program to replace the gluttony of sedentary living, becomes an unending quest for greater distances and faster times. He says that "at this point our tragic flaw emerges. Our gluttony may once again conquer us" (pp. 58-59). Willoughby (1977) says that like a pack of cigarettes, boxes for running shoes should carry a government health warning that running may be dangerous to mental health. However, there is a lack of empirical research in the area of exercise addiction.

The purpose of this discussion is not to attack running but more to incite an awareness of the dangers of overindulgence. Nor is it a certainty that all runners will become addicted or present with neurotic illnesses associated to cessation of running. Morgan considers 100 miles a week as the addiction level but notes that 50 miles a week may also be a sign of addiction. A weakness in Morgan's argument, however, is that he fails to differentiate between competitive and non-competitive runners, two groups between which motivational factors may vary greatly.

Anorexia Nervosa and Athletes

Smith (1980) observes that the pressure on young athletes to lose weight for certain sports is resulting in food aversion simulating

Anorexia nervosa. In an article titled "Excessive weight loss and food aversion simulating Anorexia nervosa in athletes", Smith says that the value of minimum fat levels in young athletes combined with a societal abhorrence of fat creates strong pressure in young sports people to reduce bodily fat levels.

In a letter to the South African Medical Journal, Norval (1980) observed that several women had presented at his office with what he described as 'Running Anorexia' characterized by high mileage and excessive weight loss. He concludes that the condition he observed was likely to become more common in the exercise oriented eighties.

In an article titled, "The Price of Perfection", Sherrye Henry (1982) reports that women running over 45 miles per week scored above the 30 point cut off score (an indicant of anorexic tendencies) on the Eating Attitude Test (EAT) (Garner and Garfinkel, 1979). She observes that the world of long distance running provides a "socially sanctioned environment" for young girls with anorexic tendencies to indulge in their obsession with food and weight loss. The pertinent question, she observes, is not why runners have to be slender, but when it is that a young girl in pursuit of the perfect runner's body becomes too thin, even anorexic. The EAT has also been used to identify AN in ballet dancers (Garner and Garfinkel, 1978).

Sours (1980) observes that AN has parallels with running and the recent growth of interest in physical fitness and Yates, Leehay and Shisslak (1983) on the basis of an interview type study have compared AN patients and runners in terms of family background, socioeconomic class, personality characteristics, denial of potentially serious debility and coming from highly exercise oriented families. They also are compared on the basis of a bizarre pre-occupation with food and an unusual emphasis on lean body mass. Sours (1980, 1981) observes that AN has increased dramatically since 1970. He notes that the provision of food was once the motivational force behind work but is now a 'given' element of life. At present the primary concern in the Western world is not to obtain enough food but rather to avoid overeating. Sours (1980) considers the major parallel between running and AN as one of a search for self-identity and to obtain control of one's life. Running is seen as dominating the runner's life much as food dominates the life of the anorexic. Alluding to Bruch's (1978) description of a "relentless pursuit of thinness", Sours says of the runner, that if he is "not gaunt, well under 150 lbs, he is out of shape, indeed fat." He further observes that the runner, like an anorexic forced to eat to gain weight, may face severe depression if he or she has to give up running for medical reasons. He notes that running, like starvation as a means of gaining self-control, may result in a repetition of the trauma that induced feelings of self-doubt and inadequacy. Later in 1981 Sours adopted a more positive stance with regard to the AN/running relationship. He states that "many long distance runners astoundingly resemble adolescents with Anorexia nervosa." He further observes that like anorexics, the runners are often hyperactive, restrict their food intake, and constantly monitor food intake and the running schedule. The runners annoy their significant others by their singular dedication to running, even to the exclusion of instinctual interests. Runners may only eat freely having first run long distances to compensate for their food consumption. He compares this to the Bulimic who gorges on food and then induces vomiting to eliminate the possibility of digestion.

Like Sours, Yates, Leehay and Shisslak (1983) note that running may be a means of establishing a self-identity and that like food avoidance in the AN patient, running may be only partially successful, perhaps even a dangerous attempt at reaffirmation of the self.

In a study using runners who logged at least 50 miles per week, Yates et al. (1983) found runners to be self-effacing, hard working, high achievers from affluent families and uncomfortable with feelings of anger. Their singular commitment to running was found to have occurred at a time of heightened anxiety, depression and identity confusion. Assuming an identity as a runner is seen as serving a means of self-fulfillment and a feeling of control over external and internal circumstances. They further note that the sense of self is enhanced as the runner acquires the elaborate rituals related to running. The desire for reduced body fat is seen as a self-perpetuating phenomenon, the runner always striving to attain new body fat percentage lows, with losses of 25% of original body weight accompanying the desire for thinness.

A parallel between running and AN, noted by Leehay, Yates and Shisslak (1983), is that of the AN and running high. They observe that increased opioid activity in runners and AN patients alike could serve as a potent reinforcer of the running or starvation habit, reducing discomfort and making either condition extremely resistant to change.

The Anorexia or running 'high' is an area in need of further research before valid comparisons can be made.

Yates, Leehay and Shisslak (1983) do, however, point out two major differences between obligatory running and classic AN. Firstly, it is noted that most anorexics are women, while the majority of obligatory runners are men and secondly, that true or primary AN usually occurs during or immediately post adolescent in girls while most of the runners in their study did not become runners until between the fourth and fifth decade of their lives. A reasonable explanation is offered in relation to different societal expectations of males and females. Attractiveness is of major importance to adolescent girls, while physical effectiveness (strength) is of more concern to the male at this time. With increasing age, the male's physical effectiveness wanes and he gains adipose tissue. The male may thus become more conscious of his lack of physical attractiveness later in life.

A final important question that must be considered is that, if one accepts that runners may calorically deprive themselves, is it possible to maintain a highly active state? The concept of hyperactivity has already been discussed in relation to AN. In an article titled, "Can we run and starve", Elrick et al. (1975) found that members of the Tarahumara tribe of Mexico trained up to 100 miles per day on a low protein, twelve to fifteen hundred calorie daily diet with no apparent ill effects.

Possible Psychological Effects of Running Literature on Runners and Would-Be Runners

An examination of popular running literature reveals several undertones important in any discussion relating AN and running.

Weight loss is consistently expressed as a virtue of a running program. Shepro and Knuttgen (1975) and Fixx (1977) observe that running will aid weight reduction whether food intake is altered or not, while Cooper (1970) considers running a means of meal replacement for weight watchers.

Related to meal replacement by running is the AN concept of the pursuit of slimness. One is continually faced in the popular running literature with the need for a thin running physique. Fixx (1977) makes the point that if you wish to run do not be satisfied with normal weight, while Sheehan (1975) describes himself as fat, although only weighing 143 lbs and having been described as "gaunt" and "cadavric".

The concept of body image is crucial in this discussion. The concepts of food avoidance and pursuit of slimness are intimately related to the concept of body image. Dr. George Sheehan (1978) is obsessed with the pursuit of the perfect form through running, while Getchell (1976) considers that people are continually looking for the magic 'formula' to achieve the ideal physique. That running improves body image has been the claim of some researchers (Lion, 1978; Fisher, and Renik, 1966; Joesting and Clance, 1979). The pertinent question in relation to a study comparing AN and running is whether or not the body image of the runner becomes distorted as the runner runs further and for an increasing number of years. To date no research has been conducted to test this hypothesis.

As AN is associated with extensive rituals related to food avoidance, then so the rigid dietary regimes and weekly schedules proposed in the popular running literature, are examples of ritual. As discussed previously, some researchers have noted the similarity of AN patients and runners in their quest for self-control (Sours, 1980, 1981; Yates et al., 1983). Such themes are prevalent in the popular running press. Sheehan (1975) and Fixx (1977) consider the fulfillment of the self and control of the self as a primary purpose of running.

There are undoubtedly similarities between AN and running. Assuming that running and AN are similar conditions from a behavioural, psychological and endocrine perspective, it remains to be ascertained whether running leads to an anorexic-like condition or whether those predisposed to AN turn to running as an expression of their need for calorie-burning activity. Anecdotal evidence suggests that the former hypothesis may be more tenable. However, until a thorough study is conducted on running involving psychological, behavioural and endocrine parameter, the link remains tenuous. Table 1 summarizes the similarities between AN and habitual long distance running.

Primary and secondary amenorrhea are important signs of AN yet secondary amenorrhea is also associated with women who run long distances and those involved in exercise necessitating the maintenance of a slim physique. Ballet dancers with extremely low percentages of body fat are often found to have amenorrhea (Frisch, Wyshack and Vincent, 1980). The problem of delayed menarche and secondary amenorrhea has been observed with exercise (Frisch, Gotz, Wilbergon and McArthur, 1981; Malina et al., 1976) and Feicht, Johnston and Martin et al., (1978) refer specifically to amenorrhea as a problem of female distance runners. Dale, Gerlach and Wilhite (1979) found that 51% of the 168 female runners in their study experienced menstrual irregularities while Frisch et al. (1981) found menarche to be delayed by almost a year on average, in a study of adolescent runners. They also found that in pre-menarchal trained runners the onset of menarche was delayed by 0.4 years for every year of training. Schwartz, Cumming and Riordon et al. (1981) found that women with regular cycles before beginning strenuous physical activity had less tendency to develop irregularity.

Mileage and severity of exercise have been cited as causes of secondary amenorrhea (Dale, Gerlach and Wilhite, 1979; Frisch et al., 1981; Feicht et al., 1978). Feicht et al. (1978) found a high correlation between training mileage and oligoamenorrhea ranging from a 6%

Table 1

SIMILARITIES BETWEEN HABITUAL RUNNING AND ANOREXIA NERVOSA

Habitual Running	Anorexia Nervosa
1. Preoccupation of runners with body image and pursuit of the ideal runner's body.	1. Distorted body image.
2. Running as a means of weight control.	2. A relentless pursuit of thinness and preoccupation with food and weight.
3. Preoccupation with caloric value of food.	3. Preoccupation with caloric value of food.
4. Denial of addiction to running.	4. Denial of illness.
5. Running: a means of controlling one's life.	5. Food avoidance: a means of achieving self-control.
6. Rituals of running.	6. Rituals of self-imposed starvation.
7. Hyperactive characteristic of habitual long distance runners.	7. Hyperactivity in AN patients. Goalless activity to burn off calories.
8. Endocrine disorder in female athletes resulting in amenorrhea. Male endocrine alterations undetermined.	8. Endocrine disorders in female and male patients. Amenorrhea in women. Reduced testosterone levels in males.

incidence in low mileage women runners to 43% in high mileage runners. Others consider the period of time over which women have been running as crucial to the development of secondary amenorrhea, the authors finding no correlation between amenorrhea with the number of miles run per week.

Fat and weight loss related to the duration of the running habit are cited as crucial factors by Speroff and Redwine (1980) while Dale, Gerlach and Wilhite (1979) found an increased incidence of amenorrhea with decreasing percentages of body fat in runners. Frisch (1977) suggests that between 10 and 15% body fat amenorrhea may occur and Frisch (1980) considered a weight loss of 2.2 kg sufficient to affect the menses. Others have also considered fat loss a factor in secondary amenorrhea (Frisch and McArthur, 1974; Frisch, 1981). Frisch (1981) in a study of runners and swimmers found amenorrheics in the group had significantly less body fat than normally menstruating runners and swimmers. It is possible that a combination of physical, hormonal, nutritional, psychological and environmental factors are responsible for secondary amenorrhea (Rebar and Cumming, 1981).

Schwartz et al. (1981) list several contributing factors from their study.

1. Amenorrheic runners were found to have pre-training histories of menstrual irregularity and thus might be pre-disposed to amenorrhea.
2. Reduced body fat and low fat/lean ratio.
3. A lower percentage of body fat in highly trained runners.
4. Abnormal LH and FSH levels and disturbance of the hypothalamic-pituitary gonadal axis secondary to weight and fat loss.
5. Stress of competition and training.

Cumming and Belcastro (1982) suggest a number of possible pathophysiological factors of exercise included changes in reproductive function.

1. Changes in lean-fat ratio.
2. Loss of weight.
3. Energy drain.
4. Dietary changes.
5. Age.
6. Pre-disposition.
7. Physical stress of training and competition.
8. Emotional stress of training and competition.
9. Acute effects of exertion.
10. Chronic effect of repeated exercise.

The nature of exercise induced amenorrhea can thus be seen to be complex. Schwartz et al. (1981) suggest that exercise induced amenorrhea is a distinct entity from hypothalamic amenorrhea (such as that found in AN). They suggest that exercise associated amenorrhea may result from peripheral changes which affect the central hormonal axis in runners whilst hypothalamic amenorrheics may suffer from a primary central axial dysfunction.

Another school of thought exists that psychological stress is a major factor (Shangold, 1981). Shangold (1981) observes that women who run more than 30 miles per week can suffer a high amount of stress attempting to co-ordinate their activities and that hormone response to stress might surpass exercise induced responses.

Endocrine Effects of Exercise in Males with Particular Reference to Male Runners

Testosterone

Most studies that have examined testosterone levels in male athletes have been concerned with the effects of acute and chronic exercise on testosterone levels or to assess the function of circulating testosterone and its affects on strength and performance. No studies to date have examined basal resting samples in long distance runners and habitual exercisers or have attempted to correlate basal and resting testosterone levels with those found in anorexic males. Some studies do report basal and pre-exercise levels as part of data collected with regard to the acute effects of exercise on hormone levels, although the resting levels are not discussed.

Basal and Pre-Exercise Levels in Male Athletes

An examination of the literature reveals that basal, pre-exercise and resting testosterone levels are within the normal 300 to 900 ng/dl range in male athletes. Sutton et al. (1973) found pre-exercise levels of 690 ng/dl in a group of swimmers and Fahey et al. (1976) 600.3 ng/dl in a group of football players. Kuoppasalmi (1980) reports normal levels of 17.3 to 26.1 nmol/litre in a group of athletes.

Studies involving long distance runners have found normal levels (Adlercreutz, 1976) and low levels (Morville et al., 1979). Morville et al. (1979) observed their runners on the day before a 100 km race had levels of 375 ng/dl (at the low end of the normal range) compared to values of 502 ng/dl in controls. However, they made no further mention of this observation.

The major problem with such studies is that few have used control groups as a comparison to the running groups and most use pre-exercise levels as opposed to the basal and resting levels. To date there are no adequate controlled studies with regard to basal testosterone levels in male long distance runners. It is therefore extremely difficult to compare endocrine dysfunctions in male AN patients and runners. No doubt this situation has arisen due to the absence of such signs as amenorrhea found in female AN patients and runners.

Effects of Acute and Chronic Exercise on Testosterone Levels

Increments in testosterone levels with exercise have been found during intense exercise whilst decreased circulating testosterone levels have been found during prolonged moderate intensity exercise. Sutton et al. (1973), Fahey et al. (1976), Kuoppasalmi (1980), and Wilkerson et al. (1980) all found that high intensity work of short duration was related to increased testosterone levels. Others have found no increase (Lamb, 1975) or a decrease during short high intensity exercise (De Lignieres, 1976). It has been suggested that apparent increases in testosterone are due to hemoconcentration (Wilkerson, Horvath and Gutin, 1980) or that increased plasma testosterone levels are correlated to pre-training levels (Fahey, Rolph et al., 1976).

Of particular relevance to this study is an examination of the testosterone response to prolonged acute exercise. Morville et al. (1979) found a significant decrease in testosterone levels from 375 to 160 ng/dl and 663 to 185 ng/dl ($p \leq 0.001$) in two groups of runners after a 100 km race and Dessypris, Kuoppasalmi and Adlercreutz (1976) found a significant decrease in testosterone levels from 23.4 to 14.1 nmol/l ($p \leq .001$) after a non-competitive marathon run. The exact mechanism by which testosterone levels decrease during endurance activities is unclear. It has been suggested that a significant rise in epinephrine could induce a drop of plasma testosterone (Morville et al., 1979). Hypercortisolemia may be responsible (Dessypris et al., 1975; Morville et al., 1979; Cumming, Quigley, and Yen, 1983) and an increased extra-hepatic or muscle consumption of androgens has been suggested (Morville et al., 1979). Aakvaag et al. (1978) suggest that reduced prolactin levels may inhibit testosterone production as prolactin may play an important role in sensitizing the testes to LH, resulting in the secretion of testosterone. However, it is generally accepted that LH levels are not responsible for a reduced plasma concentration of testosterone (Sutton et al., 1973).

Luteneizing Hormone (LH) Responses to Exercise

Alterations in plasma LH levels are not thought to be responsible for decreased testosterone levels or in testicular function during exercise (Morville et al., 1979; Dessypris et al., 1975).

Little attention, however, has been paid to resting levels in exercise studies. Dessypris et al. (1975) report levels of 15.6 Iu/l prior to a marathon run while Morville et al. (1979) found similar means levels of 1.93 ± 3.8 muI/ml in a group of runners prior to a marathon race. Once again the problem arises with regard to resting levels versus pre-exercise samples that most studies have adopted.

Increased LH levels (Remes et al., 1980) and decreased levels (Brisson et al., 1979) have been found in athletes after exercise. Remes et al. (1980) found a 25% increase in plasma LH and Brisson et al. (1979) found a decrease in LH levels after a marathon run, Schmidd et al. (1982) decreased LH levels after a cross country ski race.

Cortisol Levels and Exercise

Morville et al. (1979) found significantly increased levels of cortisol with decreased testosterone levels after a 100 km race and Dessypriss et al. (1976) found significant increases in cortisol after a marathon. Again, associated with low testosterone levels. The mechanism of increased cortisol levels may be examined. Cumming, Quigley and Yen (1983) consider that acutely increased cortisol levels cause rapid suppression of circulating testosterone levels. Insulin induced hypoglycemia was found to elevate cortisol levels which was followed by a rapid decrease in testosterone levels. They note that their findings suggest that hypercortisolism of endogenous or exogenous sources suppresses testosterone secretion by a direct action on the testes. It has been suggested that glucocorticoids may decrease testosterone function by acting on the hypothalamic-pituitary system (Bocuzzi et al., 1975; Zipf et al., 1978). The absence of any changes in LH or prolactin is seen as supporting a direct action on the testes.

The Thyroid Hormones and Exercise

Thyroxine (T_4) and Triodothyronine (T_3) and reverse Triodothyronine (rT_3) are important metabolic hormones and are particularly relevant to any discussion relating AN and habitual running. There is, however, very little data available describing resting levels in runners. Aakvaag et al. (1978) report levels of 6.8 - 7.2 μg/dl T_4 and 1.3 - 1.4 ng/ml T_3 in athletes and O'Connell et al. (1979) note levels of 26 ng/dl rT_3, 155 ng/dl T_3 and 7.0 μg/dl T_4. Kirkeby, Stromme and Bjerkaal (1977) found total T_4 levels of 7.6 μg/dl and 4.7 ng/dl free T_4 levels in their study. Balsam and Leppo (1975) found T_4 levels of 6.40 ng/dl and 131 ng/dl T_3.

Effects of Exercise on Thyroid Hormone Levels

Boyden et al. (1982) in a study of women distance runners on an endurance running program found a decrease in T_3 and rT_3 levels after the program with increased thyroid stimulating hormone responses to thyroid releasing hormone. The authors consider this indicative of mild thyroidal impairment. O'Connell et al. (1979) found that prolonged moderate exercise on a bicycle resulted in raised rT_3 and T_4 levels but decreased T_3 levels. The authors observe that such activity induces a state equivalent to acute starvation and that raised rT_3 levels parallel the condition found in starving people (Vagenakis et al., 1975). It was noted that a positive correlation ($r = 0.95$) of raised rT_3 levels and free fatty acids (FFA) was indicative of a relationship of thyroid hormones and lipid metabolism. Kirkeby et al. (1977) found a significant increase in T_4 after exercise while Balsam and Leppo (1975) noted decreased T_4 levels after exercise.

Irvine et al. (1967) found that T_4 increased initially during endurance exercise in horses but eventually fell due to increased T_4 degradation during exercise, using the $T_4{}^{125}I$ tracer method. Later, Irvine (1968) found a similar increased turnover of T_4 in humans, thus offering an explanation for reduced T_4 levels during exercise with concomitant increases in T_3.

CHAPTER III

METHODOLOGY

Subjects

Sixty club runners from the Edmonton area were recruited and divided into 3 groups according to mileage (Group 1: 20-39 miles per week, Group 2: 40-69 miles per week, Group 3: 70 plus miles per week).

Twenty university runners were selected who ran at least 20 miles per week.

Twenty non-exercising controls were chosen for their lack of any regular exercise habits. Controls were selected on the basis of a complete absence of participation in or interest in physical activity.

Age of Subjects

Habitual Runners	30 - 55
University Runners	19 - 25
Controls	19 - 25

Equipment

1. Questionnaires:
    - (a) Personal Data Questionnaires: to ascertain details with regard to profession, educational background and achievement and parental encouragement for achievement.
    - (b) Eating Attitude Test (Garner and Garfinkel, 1979)
    - (c) Jackson Personality Inventory (Jackson, 1962)
    - (d) Running Commitment Questionnaire

2. Body Image Drawing Test (Askevold, 1975):

   Required 2 marking pens and plain marker approximately 5 feet by 4 feet placed on a vertical surface.

3. Radio-Immuno Assay Kits and Vacuo-Tubes:

   For the collection of resting blood samples.

Summary of Procedures

1. One hundred subjects were selected according to previously discussed criteria.
2. Subjects were asked to attend a testing session requiring a one-and-one-half hour to two hour commitment.
3. Subjects were asked to attend between 4:00 p.m. and 7:00 p.m. in the evening for the purposes of collating uniform basal plasma hormone samples.
4. On arrival the tester explained to the subjects the nature of tests involved and expressed the need for honesty in responses to questionnaires. The purpose of the study was, however, to remain concealed until after the study was completed such that results would not be biased by 'on guard' responses.
5. Identity numbers were assigned at random.
6. A maximum of 15 subjects were interviewed at any one time.
7. There was no selected group testing in order to eliminate a group bias effect.
8. Having explained the constraints under which the tests would occur, each subject was asked to:
   (a) Read and consign a consent form outlining risk ractors and experimental procedure.
   (b) Fill out a set of selected questionnaires.

(c) Partake in a simple drawing test to assess body image.

(d) Give a 20 ml blood sample, taken by a qualified physician, for the purposes of assessing plasma hormone levels.

9. Data collected was analyzed thus. Questionnaire data related to running commitment and demographic background was examined via distributors and bar groups.

A one-way analysis of variance was implemented in order to ascertain differences between groups. A post hoc test (Scheffe) was implemented to identify significant differences between pairs of groups.

Correlations between Eating Attitudes as defined by an EAT score, body image, as defined by Body Image Perception index scores and Personality as defined by 1b personality sub-type scores. A correlation matrix was calculated between these variables and hormone levels.

10. Analysis Grouping: Mileage effect on the dependent variables was ascertained via progressive composite grouping. The initial analysis comprised 5 groups.

Group

1) University runners
2) 70 miles per week plus runners
3) 40-69 miles per week
4) 20-39 miles per week
5) Control group (non-exercise group)

The university group was then divided according to mileage into the 3 runner groups. This gave 4 groups.

Group

1) 70 plus miles per week
2) 40-69 miles per week
3) 20-39 miles per week
4) Control group

Groups one and two were then amalgamated to form one 40 miles per week plus group.

Group

1) 40 plus miles per week

2) 20-39 miles per week

3) Control group

Examination of Questionnaires and Test Protocols

1. Running Commitment Questionnaire: This included questions with regard to mileage covered per week, time at which running became a regular event or habit. If the respondent was unsure an approximation was requested of him. Other questionnaire items elicited responses with regard to feelings on running and on having to miss a run.
2. Demographic Questionnaire: This questionnaire was designed to collect information on subject ID number, height, weight, age, subject achievement, motivation, family and economic background, parental encouragement and professional status.
3. Eating Attitude Test (EAT): This test was developed and validated for evaluation of a broad range of target behaviours and attitudes in Anorexia nervosa (Garner and Garfinkel, 1979). The test had a reported reliability coefficient of 0.79 (Garner and Garfinkel, 1979). A score of 30 plus on the test was taken to be a cut-off score and indicative of anorexic traits and tendencies. The test does, however, have a limitation in that there is no check for a denial factor; denial of illness being a common condition in anorexic patients. An emphasis on integrity was essential on administration of the test in

light of this limitation.

This test was chosen over a similar 22 item scale developed by Slade (1973) because the Eating Attitude Test measures a broad range of behaviours while the former concentrates on only 3 areas of Anorexia.

4. The Jackson Personality Inventory (JPI): The Jackson Personality Inventory (JPI) is an improvement of the Personality Research From (Jackson, 1962). Its purpose is to provide in one convenient form, a set of measures of personality reflecting a variety of interpersonal, cognitive and value orientation likely to have important implications for a person's functioning.

The questionnaire is designed for a population of average or above average ability and comprises 320 True-False statements, incorporating 16 scales, each containing 20 statements, scoring is by template.

Administration time is approximately 40-50 minutes and the form has language simplified to high school level to ensure universal comprehension of test items. Each scale comprises ten true keyed and ten false keyed statements in order to:

a) minimize the role of acquiescence, and

b) to permit definition of each pole of a bipolar scale dimension with positively worded content.

The JPI is designed for use either by qualified psychologists or an unskilled user if under supervision of a psychologist or where adequate psychological consultation is available.

The JPI comprises the following scales:

1. Anxiety
2. Breadth of Interest
3. Complexity
4. Conformity
5. Energy Level
6. Innovation
7. Interpersonal Effects
8. Organization
9. Responsibility
10. Risk Taking
11. Self-Esteem
12. Social Adroitness
13. Social Participation
14. Tolerance
15. Value Orthodoxy
16. Infrequency

Although easy to administer, the tester had to be aware of questionnaires answered in a manner other than with the degrees of integrity needed to achieve valid results. Three areas requiring special attention were:

i) non-purposeful responding,

ii) faking and motivated distortion, and

iii) membership of a population different from those on which norms were based.

Expected Results: According to the hypothesis of anorexic behaviours, the author expected to find high Anxiety scores and low scores on the Conformity, Interpersonal Effect, Self-Esteem, Tolerance of Others, Value Orthodoxy and Responsibility and Obligation to Others scales.

Body Image Test

The test used was that devised by Askevold (1975). The author chose this method over that of Reitman and Cleveland (1964) and Slade and Russell (1973) because the method involved subject participation in producing a drawn body image whilst the latter two involved visual size perception in which the subject was not directly involved in image presentation.

Protocol

1. Subject stood facing the paper at arm's length.
2. Subject was given two marker pens at arm's length.
3. The subject was instructed to hold his arms in a fully extended position but not to touch the paper until instructed to do so and not to let the arms fall below a horizontal plane.
4. The subject was instructed to imagine that he was looking into a mirror.
5. The tester then touched the subject on the head, shoulders, waist and on the hips, with both hands. The subject was asked by the tester to mark the points touched on the paper to produce a body profile. To establish the degree of reliability of the test, the subject was asked to perform the test twice.
6. Measurements of the actual subject were taken after the second image marking session using anthropometric calipers.
7. Heights and widths as perceived by the individual were measured directly from the paper.
8. Body Perception Index (Slade and Russell, 1973) was calculated by the following formula:

$$\frac{\text{Perceived Size}}{\text{Real Size}} \times \frac{100}{1}$$

A score of over 100 indicated an overestimation of body width and a score of less than 100 indicated an underestimation of bodily dimensions.

Hormone Analysis

A blood sample was taken from each individual by a qualified physician. Blood samples were taken between 4:00 p.m. and 7:00 p.m. in the evening to ensure basal plasma levels of testosterone.

Testosterone, cortisol, prolactin, luteinizing hormone and follicle stimulating hormone levels were assessed by radio-immuno assay. Sex hormone binding globulin levels were assessed via a modification of the method of Rudd et al. (1974). Free and non-SHBG bound testosterone were calculated using an equation based on the law of mass action and assuming two binding systems. A full description of the assay procedures can be found in Appendix 10.

CHAPTER IV

RESULTS

PART I

A. Questionnaire Analysis

The demographic data and running commitment questionnaires were analyzed by frequencies and percentages and are reported below. Summary tables of data appear in Appendices 2 and 3.

Demographic Data

Age Groups of Runners

University runners ranged from 18-25 ($\bar{X} = 21.8 \pm 0.94$), 70+ miles per week runners from 27-56 ($\bar{X} = 38.8 \pm 4.08$), 40-69 miles per week runners from 18-50 ($\bar{X} = 36.3 \pm 2.33$) and 20-39 miles per week runners from 25-49 ($\bar{X} = 39.7 \pm 1.74$). Controls ranged in age between 18-42 ($\bar{X} = 27.68 \pm 1.35$). Differences in age ranges were compounded by subjects failing to show for testing.

Heights and Weights of Runners

Heights and weights of runners and controls were utilized to calculate a ponderal index for each group. Analysis of variance revealed significant differences among the groups on the ponderal index scores at the 0.01 level, however post hoc analysis revealed no significant difference between any groups at any analysis level, suggesting that within group or error variance accounted for variation among subjects and not a group effect. Means, standard deviations and standard errors may be found in Appendix 2(b).

Professional Status of Groups

Responses were categorized as either Professors/Teachers, Business/Management, Medical (M.D., Nurse), Laboratory Technicians, Legal, Government, Journalism, Engineering, Students and non-professional manual occupations. All university runners fell under the Student category as well as 3 of the total number of runners (6%). Eight runners (16.3%) fell in the Professor/Teacher category, 12 (25.5%) in the Business/Management category, 1 (2%) in the Medical category, 4 (8%) in the Lab Technician category, 3 (6%) in the Legal category, 2 (4%) in the Government category, 2 (4%) in Journalism and 1 (2%) in Civil Engineering. Only 2 runners (4%) were engaged in other manual and non-professional occupations. Controls were almost exclusively students (n = 17, 94%) and 1 fell in the Professor/Teacher category.

Overall, 96% of the runners fell into Professional or Technical service occupations with only 4% as students or non-professional occupations.

Income Brackets

Income brackets reflected the above situation. Fifty-three percent of runners (n = 26) fell in the $30,000 plus category, 14% (n = 7) in the $25,000-30,000 category, 4% in the $20,000-25,000 and $15,000-20,000 categories. This excluded the university runners who fell exclusively into the less than $15,000 per year category. Sixty-seven percent of the runners interviewed were thus found to earn in excess of $25,000 per year. Controls were found to fit mainly into the less than $15,000 per year category (72%).

Educational Achievement Levels

Of the 70+, 40-69 and 20-39 miles per week running groups, 85% were found to have achieved Bachelor degree status or better. Fifteen percent of the runners had achieved a high school education and no further qualifications. University runners, by virtue of being students were primarily at the undergraduate level (40%), although 30% had a Bachelor degree and 10% a Masters degree. Controls were again primarily students at the undergraduate level (44% high school graduate level). Thirty-three percent had existing Bachelor degrees and 16% had Masters level degrees. Five percent had a Ph.D. or higher.

External Achievement Motivation

Of the total number of runners (n = 49), 86% were found to have been strongly encouraged to achieve at school. Fourteen percent responded that they had not been encouraged to achieve at school.

Similarly, the majority of the control group (94%) had been encouraged to achieve highly.

Number of Years Engaged in Serious Running

University runners reported a mean of 6.3 years, 70+ miles per week group, 13.1 years, 40-69 miles per week, 15.2 and 20-39 miles per week group, 11.3 years. The grand mean for years engaged in running was 11.4 years.

B. Analysis of Running Commitment Questionnaire

Mileage Categories

According to previously defined mileage categories, runners could be split into 3 groups: 70+, 40-69 and 20-39 miles per week. The following frequencies were obtained:

Mileage Category	Frequency	University Runners
20-39 mpw	18	3
40-69 mpw	15	2
70+ mpw	6	4

Number of Runs Per Week

The mean number of runs per week was calculated for each group. University runners were found to run an average 8.5 times per week, 70+ runners, 9.5 times per week, 40-69 miles per week runners, 6.9 times and 20-39 miles per week runners, 5.0 times per week. Differences in runs per week frequency are due to mileage covered.

Of the total number of runners (n = 49), 16 ran 5 times per week (34%), 12 ran 6 times per week (26%) and 11 ran 7 times per week (24%). Those running between 5 and 7 times per week accounted for 84% of the runners. Sixteen percent ran more than 7 times per week including 2 runners (4%) who ran at least 14 times per week.

Competition

Of the total number of runners 3 were found to compete at every opportunity (6%), 24 (48%) competed frequently and 21 (43%) competed occasionally; only one runner (2%) was found not to compete at all.

Running and Time of Day

Of the total number of runners, 26% (n = 13) were found to run prior to breakfast, 35% (n = 17) between breakfast and lunch, 61% (n = 30) between lunch and supper and 60% (n = 3) after supper. Percentages are given as a comparison of each to group total in that percentages include 22% of the runners who ran at more than one of these times.

In the university group 5 ran more than once per day, with 1 runner running 3 times. In the 70+ miles per week group 5 also ran more than once per day and in the 40-69 miles per week group 7 runners ran more than once per day. Two ran more than once per day in the 20-39 miles per week group. This represents 50% in the university group, 83% of the

70+ miles per week group, 47% in the 40-69 miles per week group and 11% in the 20-39 miles per week group.

Of the total number of runners, 11 ran more than once per day. This represents 22% of the total number of runners. However, while only 2 runners in the 70+ group responded that they ran more than once per day, 3 in fact indicated that they ran more than once via other category responses to this question.

Frequency of Missing Meals

Of the total number of runners, 8% missed breakfast (n = 4), 26% missed lunch (n = 13) and 4% missed supper (n = 2) regularly in order to run. Sixteen percent (n = 8) responded that they never missed a meal while 55% (n = 27) responded that they occasionally missed a meal. Eight percent (n = 4) responded that meals were missed frequently in order to run.

Running and Lateness for Work

Sixty percent (n = 29) of the runners responded that running never made them late for work while 35% (n = 17) said that running occasionally made them late. Three runners representing 6% of the total number responded that running made them late for work frequently.

Number of Meals Eaten per Day

The majority of the runners as a group (n = 29, 60%) consumed 3 meals a day. Thirty-four percent (n = 17) consumed 2 per day while only 6% (n = 3) consumed only one meal per day. The majority of the 20-39 and 40-69 miles per week runners consumed three meals per day (61% and 67% respectively) although 50% of the 70+ runners and university runners consumed only 2 meals per day.

Running and Domestic Responsibility

Ten out of the total of 49 runners (20%) responded that running never interfered with domestic responsibilities. Thirty-five runners representing 71% of the total number of runners answered that running occasionally interfered with domestic responsibilities while 4 runners (8%) admitted that running often interfered with domestic matters.

Ninety percent of the university runners responded occasionally, while 66% of the 70+ group responded occasionally, 33% very often. In the 40-69 miles per week group 60% responded occasionally with only 1 runner stating (6%) reporting often. Five 40-69 miles per week runners representing 33% of the group responded that running never interfered with domestic responsibility.

Seventy-two percent of the 20-39 miles per week runners responded that running occasionally interfered while only 1 reported frequent interference. Twenty-two percent (n = 4) responded that running never interfered with daily domestic responsibility.

The Significance of Running in Daily Life

Forty-seven percent of the runners (n = 23) reported that running was a significant part of their daily lives as a primary response to this question. Twelve runners (25%) reported that running had a fitness significance to them while only 2 runners (4%) considered running an important means of weight control. Fourteen percent responded that running was for enjoyment while 4% reported that running fulfilled a hobby function.

Fifty percent of the university and 20-40 miles per week running groups considered running fundamental to their existence compared to 26% of the 40-69 miles per week group. Notably, the 70+ miles per week runners were exclusively found in the fundamental importance category.

Response to a Doctor's Orders of Cessation of Running during Injury

Of the total number of runners 71% responded that they would partially take a doctor's advice and rest for a week prior to trying out the leg. Six runners, representing 12%, said they would completely ignore the physician while 7 runners (14%) said they would obey the doctor to the letter. The majority of runners in all groups fell in the 'partially ignore' category and 'ignore orders' respondents were distributed evenly throughout the groups.

Emotions Related to Loss of the Daily Run

Of the total number of runners 53% (n = 26) reported that they would be mildly regretful if a daily run was missed. Thirty-four percent (n = 17) responded that they would be anxious, while only 1 runner (2%) said he would be extremely anxious if the run was missed. Eight percent of the runners (n = 4) reported an 'indifferent' response. Sixty percent of the university runners reported 'mildly indifferent' compared to 16% of the 70+ miles per week runners, 46% of the 40-69 miles per week group and 67% of the 20-39 miles per week group. Sixty-six percent of the 70+ miles per week group reported 'anxious' compared to 46% of the 40-69 miles per week group and 27% of the 20-39 miles per week group.

Need to Run Following Ingestion of Food

Seventy-seven percent of the total number of runners reported that they never felt the need to run directly after a meal while 20% responded that this need was felt 'sometimes'. No runners responded that they always felt the need to run directly after a meal.

Immediate Reactions to Knowledge of Need to Cease Running

Twenty runners (40%) said they would hunt for an alternate form of exercise while 14 runners (20%) responded with feelings of extreme depression, loss and anxiety. Eight runners (16%) said they would ignore the physician totally while only 2 runners (4%) had any concern in relation to gains in weight. One runner expressed an immediate concern over loss of fitness. The above categories represented those into which responses fitted most readily.

PART II

Establishing Reliability of the Body Image Test

A Pearson product moment correlation was calculated between each repeated body image measure. Correlations between shoulder, waist, hip and height measures were found to be high, positive and significant in all cases ($p < 0.05$). Shoulder 1 and shoulder 2 produced a r of 0.8096, waist 1 to waist 2, $r = 0.8176$, hip 1 to hip 2, $r = 0.7646$, height 1 to height 2, $r = 0.6514$ and mean 1 to mean 2, $r = 0.8314$.

Statistical Tests of Body Image, Personality and Hormone Data

For all of the above a one-way analysis of variance (ANOVA) was computed ($\alpha = 0.05$). Each analysis was repeated over 5, 4 and 3 groups. Groups were divided as follows:

5 Group Analysis:	Group 1	University runners
	Group 2	70 miles per week club runners
	Group 3	40-69 miles per week club runners
	Group 4	20-39 miles per week club runners
	Group 5	Non-exercising controls

4 Group Analysis:	Group 1	70 miles per week plus
	Group 2	40-69 miles per week
	Group 3	20-39 miles per week
	Group 4	Controls
3 Group Analysis:	Group 1	40 miles per week plus
	Group 2	20-39 miles per week
	Group 3	Controls

Following a significant ANOVA, post hoc Scheffé between group comparisons were computed to ascertain significant differences between pairs of groups.

Significant ANOVAs will be represented by (F Prob =) and Scheffé degrees of significance at either the $p < 0.1$ or $p < 0.05$ level. Means will be given plus the standard error of the mean ($\overline{X}$, $\pm$ SEM).

Results are presented as summary tables in the appendices.

Body Image Test Results

5 Group Analysis

Analysis of variance was significant (F prob = 0.0409) for the shoulder 1 measure. Group 1 ($\overline{X} = 88.79 \pm 4.45$) was significantly different than Group 2 ($\overline{X} = 118.91 \pm 12.89$, $p < 0.1$). No other measure was significantly different on the ANOVA. However, Group 3 overestimated waist width by $\overline{X} = 36.96 \pm 10.15$ and $\overline{X} = 31.5 \pm 10.39$ compared to controls, $\overline{X} = 17.02\% \pm 6.87$ and $\overline{X} = 5.38\% \pm 5.95$. Group 2 overestimated body width by $\overline{X} = 23.3\% \pm 7.02$ and $\overline{X} = 21.1\% \pm 9.75$. Grand means of 119.3 ± 3.55 and 117.69 ± 3.66 for waist width indicated a general tendency for all subjects to overestimate waist width. Runners over 40 miles per week overestimated to a greater degree than low mileage runners (20-39 miles per week) and controls.

Hip measures were also overestimated in all individuals while shoulder and height measures were generally underestimated. An exception was found in Group 2 runners who overestimated shoulder width by $\overline{X}$ = 18.9% ± 12.89 and $\overline{X}$ = 11.7% ± 10.94 on measures 1 and 2. Height was persistently underestimated by approximately 24% over all groups. A trend of over-estimation of body dimensions in terms of shoulder, hip and waist width is apparent in runners over 40 miles per week. University runners were noticeably more accurate in their perceptions.

4 Group Analysis

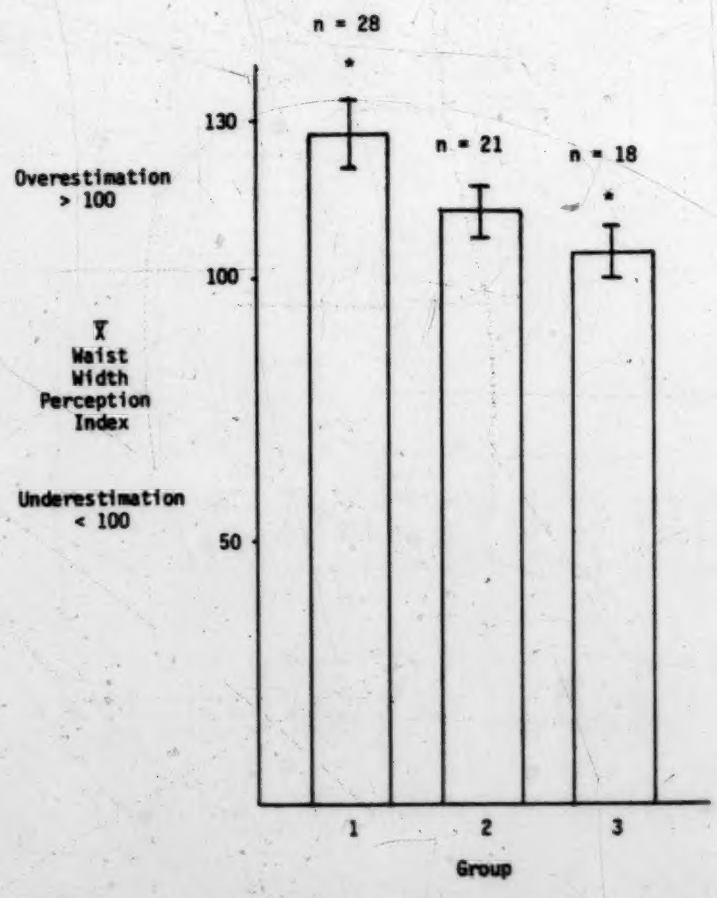
Analysis of variance and post hoc Scheffé test revealed no significant differences among the groups or pair of groups. All groups showed a tendency towards overestimation of waist and hip width. Group 3 ($\overline{X}$ = 97.99 ± 4.22, $\overline{X}$ = 97.29 ± 4.21) and controls (Group 4) ($\overline{X}$ = 95.60 ± 3.48, $\overline{X}$ = 92.58 ± 3.75) underestimated shoulder width. All groups underestimated height by between 20 and 31%.

Waist width was the most overestimated measure. Group 2 overestimated by $\overline{X}$ = 29.73% ± 9.31 and $\overline{X}$ = 30.50% ± 9.14 compared to controls, $\overline{X}$ = 17.02% ± 6.87 and $\overline{X}$ = 5.38% ± 5.95.

Group 2 showed the largest mean overestimation of body size, $\overline{X}$ = 19.40% ± 6.54, $\overline{X}$ = 16.56% ± 5.4. The grand mean for estimation of height over all groups was $\overline{X}$ = 74.34% ± 1.49 and $\overline{X}$ = 74.4% ± 1.69, representing a group tendency for an approximate underestimation of 25% of height.

3 Group Analysis

The ANOVA was significant for waist 2 measure (F Prob = 0.0443). A post hoc Scheffé test revealed a significant difference between Group 1 ($\overline{X}$ = 127.32 ± 6.24) and controls ($\overline{X}$ = 105.38 ± 5.15, $p < 0.05$)(Figure 1).



* Group 1 vs 3 significantly different, $p < 0.05$

Figure 1. Mean estimation of waist width: 3 group analysis

This represents an overestimation of waist width of 27.32% in Group 1 compared to 5.38% in controls. Group 1 overestimated waist width in measure 1 by $\overline{X} = 27.28\% \pm 6.21$ compared to $\overline{X} = 17.02\% \pm 6.87$ in controls. This difference was not significant. All groups showed a tendency to overestimate waist width. Shoulder estimates were most accurate in all groups. The range of underestimation of height in groups was between 23.3 and 28.5%. All groups showed a tendency to underestimate height.

Mean scores for groups revealed that Group 1 runners overestimated bodily dimensions by $\overline{X} = 17.4\% \pm 4.69$ and $\overline{X} = 15.85\% \pm 4.47$ compared to controls, $\overline{X} = 10.84 \pm 4.59$ and $\overline{X} = 2.9\% \pm 4.33$.

Summary tables of means, standard deviations and standard errors appear in Appendix 4.

Eating Attitude Test

5, 4 and 3 Group Analysis

Analysis of variance revealed no significant differences on EAT scores for any groups or between any pairs of groups on either the 5, 4 or 3 group analysis. A summary table of results appears in Appendix 5.

Jackson Personality Inventory

5 Group Analysis

The ANOVA for energy level was significant (F Prob = 0.0263). Post hoc analysis revealed that Group 4 ($\overline{X} = 55.17 \pm 1.90$) scored significantly higher than controls ($\overline{X} = 45.72 \pm 2.28$) on energy level ($p < 0.1$). The ANOVA for responsibility was significant (F Prob = 0.0006). Group 1 ($\overline{X} = 43.5 \pm 3.2$) was found to score significantly lower than Groups 2 ($\overline{X} = 58.3 \pm 2.40$), 3 ($\overline{X} = 56.86 \pm 1.97$) and 4 ($\overline{X} = 43.5 \pm 3.2$) ($p < 0.05$) although there was no significant difference between Group 1 and controls. The ANOVA for infrequency was significant (0.0269) although post hoc analysis revealed no significant differences between pairs of groups.

No other personality variables were found to be significantly different among the groups.

4 Group Analysis

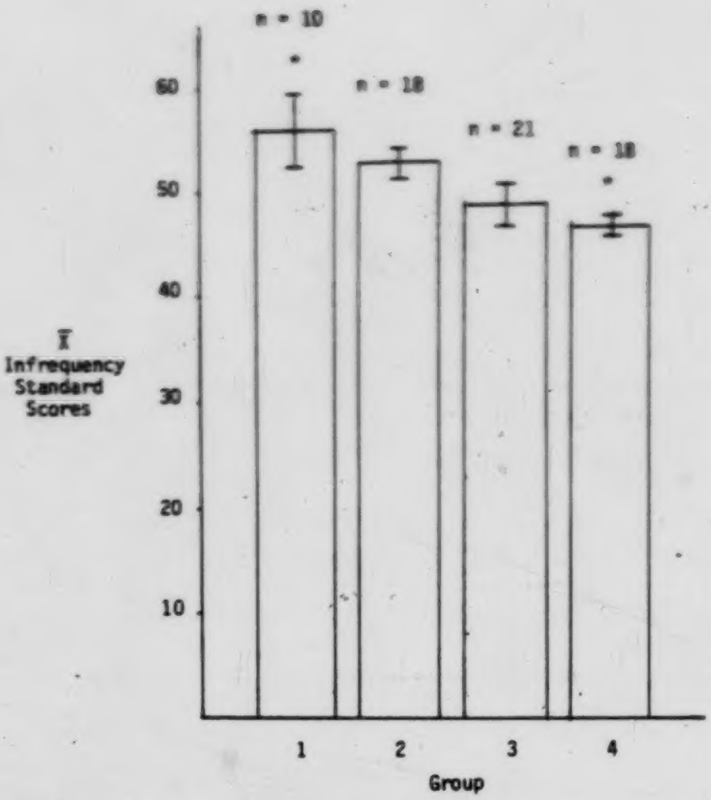
Although the ANOVA approaches significant (F Prob = 0.066) for energy level, distribution of university runners among the groups eliminated the significant difference between low mileage runners and controls. The ANOVA for infrequency was significant (F Prob = 0.0143). Post hoc analysis showed that Group 1 ($\overline{X} = 56.4 \pm 3.34$) scored significantly higher on infrequency scores than Group 4 (controls) ($\overline{X} = 47.0 \pm 1.02$, $p < 0.05$) (Figure 2).

ANOVA and post hoc analysis revealed no significant differences among the groups or pairs of groups on any other variables.

3 Group Analysis

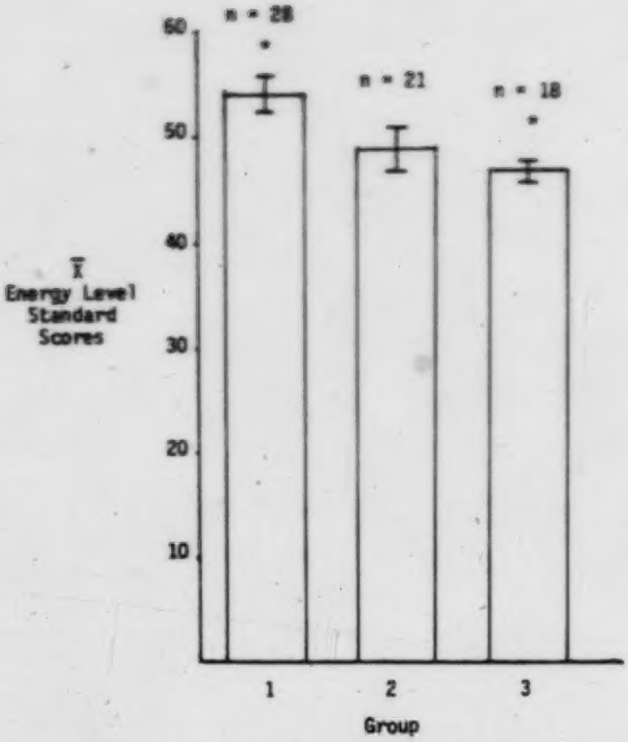
The ANOVA for energy level was significant (F Prob = 0.0362). Post hoc analysis showed that Group 1 ($\overline{X} = 52.92 \pm 1.71$) scored significantly higher than Group 3 ($\overline{X} = 45.72 \pm 2.28$, $p < 0.1$) on energy level (Figure 3). The ANOVA for infrequency was significant (F Prob = 0.0088). Post hoc analysis revealed that Group 1 ($\overline{X} = 54.21 \pm 1.54$) scored significantly higher than Group 2 runners ($\overline{X} = 49.14 \pm 2.12$, $p < 0.1$) and Group 3 (controls) ($\overline{X} = 47.0 \pm 1.03$, $p < 0.05$) (Figure 4).

ANOVA revealed no significant differences among the groups on any other personality variable. Summary tables of means, standard deviations and standard errors for the 5, 4 and 3 group analysis appear in Appendix 6.



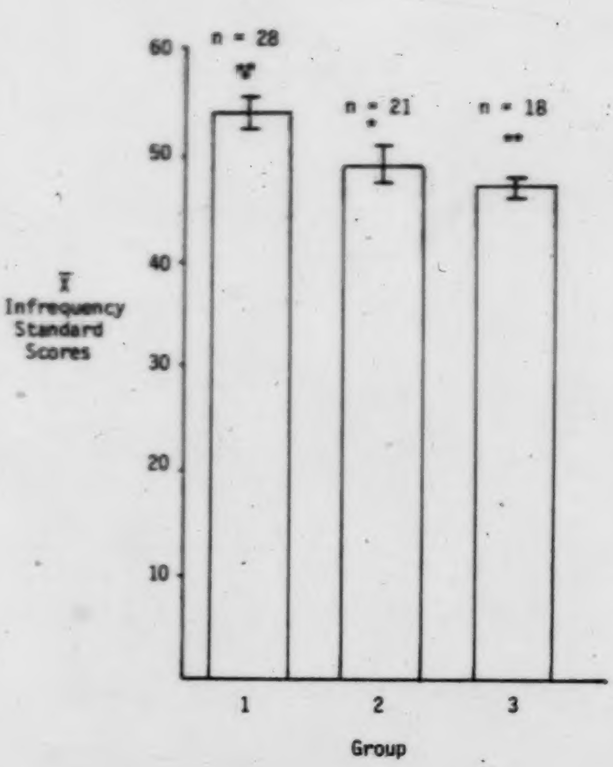
* Group 1 vs 4 significantly different, $p < 0.05$

Figure 2. Personality measures: mean infrequency scores: 4 group analysis



* Group 1 vs 3 significantly different, $p < 0.1$

Figure 3. Personality measures: mean energy level, 3 group analysis



* Group 1 vs 2 significantly different, $p < 0.1$
** Group 1 vs 3 significantly different, $p < 0.05$

Figure 4. Personality measures: mean infrequency scores, 3 group analysis

Hormone Analysis

5 Group Analysis

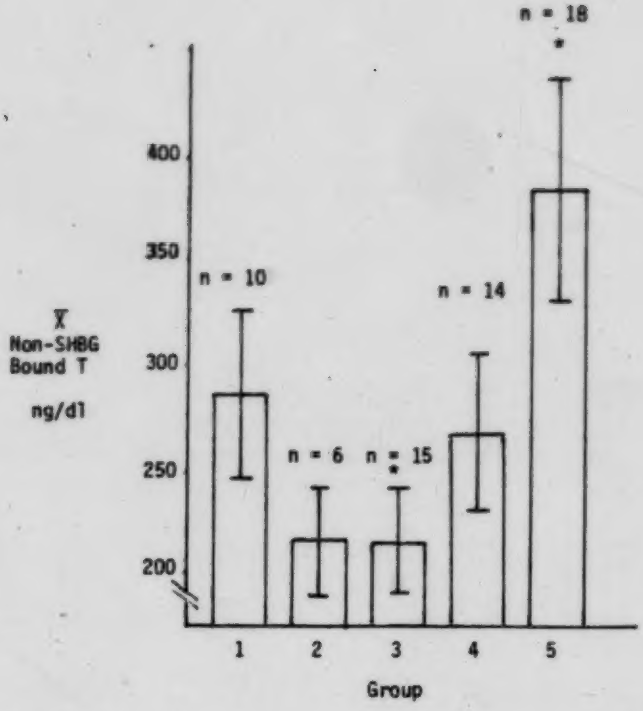
The ANOVA for SHBG was significant among the groups (F Prob = 0.0060). Post hoc analysis via a Scheffé test revealed that Group 1 ($\bar{X} = 4.83$ ng/dl ± 0.18) had significantly lower SHBG levels than Group 3 runners ($\bar{X} = 5.96$ ng/dl ± 0.25, $p < 0.05$) and Group 3 runners had significantly higher levels than controls ($\bar{X} = 5.11$ ng/dl ± 0.018) but only at the 0.1 level of significance.

A significant ANOVA was computed for non-SHBG bound T (F Prob = 0.0195). Post hoc Scheffé tests revealed significantly lower non-SHBG bound T levels in Group 3 runners ($\bar{X} = 216.16$ ng/dl ± 24.25) than Group 5((controls) ($\bar{X} = 387.31$ ng/dl ± 53.51, $p < 0.05$) (Figure 5).

The ANOVA for T levels was significant (F Prob = 0.0473). However, no between group differences were significant in post hoc analysis. Runners as a group appeared to have considerably lower T levels than controls. No other ANOVA was significant.

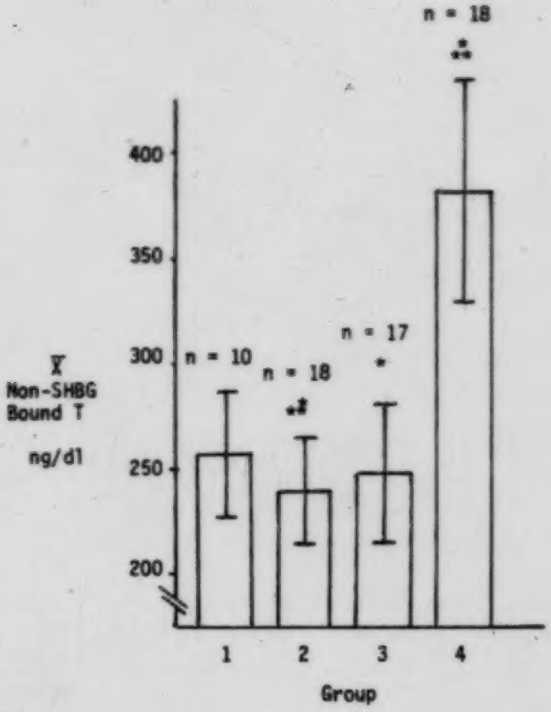
4 Group Analysis

The ANOVA for non-SHBG bound T was significant among the groups (F Prob = 0.0181). Post hoc Scheffé tests revealed that Group 2 ($\bar{X} = 240.02$ ng/dl ± 24.68) had significantly lower non-SHBG bound T levels than Group 4 (controls) ($\bar{X} = 387.31$ ng/dl ± 53.51, $p < 0.05$). Group 3 runners ($\bar{X} = 248.62$ ng/dl ± 31.19) had significantly lower non-SHBG bound T levels than controls but only at the 0.1 level of significance (Figure 6). The ANOVA for Free T was significant (F Prob = 0.0257). Post hoc analysis showed that Group 3 runners had significantly lower Free T levels ($\bar{X} = 9.08$ ng/dl ± 1.13) than Group 4 controls ($\bar{X} = 15.15$ ng/dl ± 2.41, $p < 0.05$) (Figure 7).



* Group 3 vs 5 significantly different, $p < 0.05$

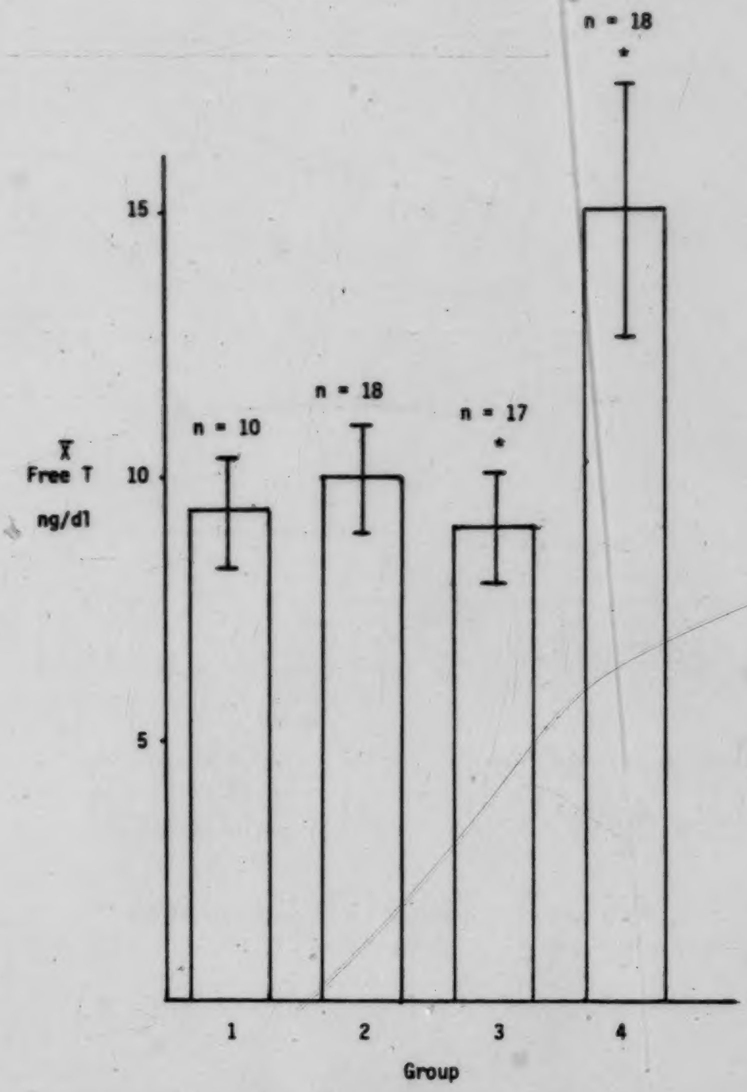
Figure 5. Mean non-SHBG bound T levels, 5 group analysis



** Group 2 vs 4 significantly different, $p < 0.05$

* Group 3 vs 4 significantly different, $p < 0.1$

Figure 6. Mean non-SHBG bound T levels, 4 group analysis



* Group 3 vs 4 significantly different, $p < 0.05$

Figure 7. Mean free testosterone levels, 4 group analysis

The ANOVA for LH levels was significant (F Prob = 0.05) for runners as a group versus controls although post hoc analysis revealed no differences between groups. Examination of the data revealed a considerable difference between Group 3 runners ($\overline{X}$ = 6.71 mIu/ml ± 0.73) and controls ($\overline{X}$ = 10.10 mIu/ml ± 1.04). Group 3 was also noticeably lower than the high mileage Group 1 ($\overline{X}$ = 10.56 mIu/ml ± 1.32) on the LH measure.

The ANOVA for prolactin was significant (F Prob = 0.01). However, post hoc analysis revealed no significant differences between groups. Although the ANOVA for TSH approached significance (F Prob = 0.06), there is no clear pattern with regard to TSH levels among the group.

No other ANOVAs were significant among the hormones measured.

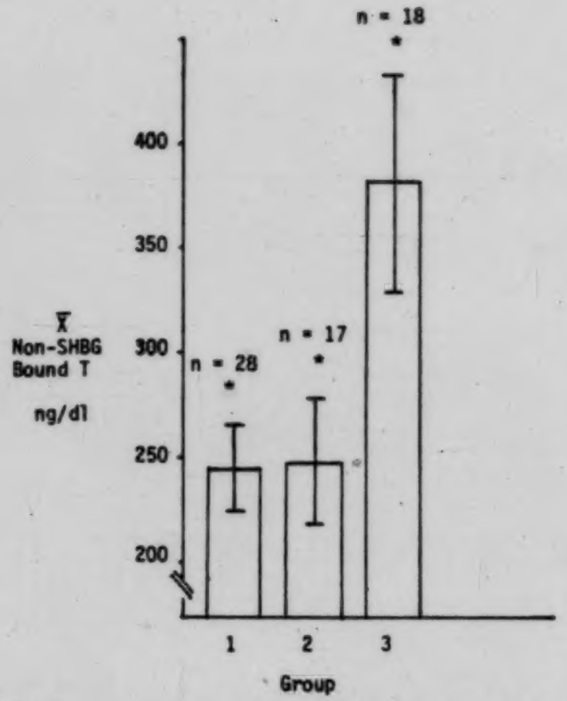
3 Group Analysis

ANOVA was significant for LH levels (F Prob = 0.034). Post hoc analysis revealed significant differences only at the 0.1 level, between Group 2 runners ($\overline{X}$ = 6.71 mIu/ml ± 0.73) and Group 1 ($\overline{X}$ = 9.63 mIu/ml ± 0.85) and Group 2 and controls ($\overline{X}$ = 10.10 mIu/ml ± 1.04, $p < 0.1$).

The ANOVA for non-SHBG bound T was significant (F Prob = 0.006). Group 1 had significantly lower non-SHBG bound T levels ($\overline{X}$ = 246.44 ng/dl ± 18.99) than controls ($\overline{X}$ = 387.31 ng/dl ± 53, $p < 0.05$). Group 2 runners ($\overline{X}$ = 248.62 ng/dl ± 31.19) had significantly lower non-SHBG bound T levels than controls ($p < 0.05$) (Figure 8).

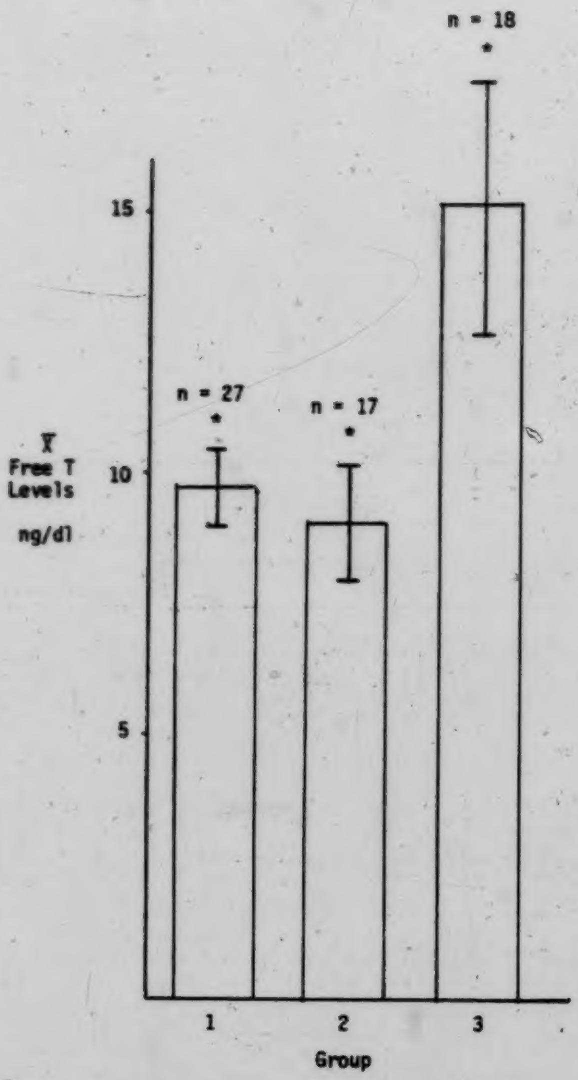
ANOVA for Free T was significant (F Prob = 0.0095). Group 1 was found to have significantly lower Free T levels ($\overline{X}$ = 9.76 ng/dl ± 0.76) than Group 3 (controls) ($\overline{X}$ = 15.15 ng/dl ± 2.41, $p < 0.05$) and Group 2 runners had significantly lower Free T levels ($\overline{X}$ = 9.08 ng/dl ± 1.13) than controls ($p < 0.05$) (Figure 9).

The ANOVA for TSH was significant among groups (F Prob = 0.03).



* Group 1 vs 3 significantly different, $p < 0.05$
* Group 2 vs 3 significantly different, $p < 0.05$

Figure 8. Mean non-SHBG bound T levels, 3 group analysis



* Group 1 vs 3 significantly different, $p < 0.05$

* Group 2 vs 3 significantly different, $p < 0.05$

Figure 9. Mean free testosterone levels, 3 group analysis

Group 1 runners had significantly lower TSH levels ($\overline{X}$ = 4.85 uU/ml ± 0.88) than Group 2 runners ($\overline{X}$ = 5.93 uU/ml ± 0.56) and Group 2 runners, significantly higher TSH levels than controls ($\overline{X}$ = 4.79 uU/ml ± 0.18). However, both comparisons only reached significance at the 0.1 level on the Scheffé comparison.

The ANOVA for prolactin was significant ($p < 0.05$) and post hoc analysis showed that Group 1 runners ($\overline{X}$ = 6.07 ng/ml ± 0.83) had significantly lower prolactin levels than controls ($\overline{X}$ = 8.01 ng/ml ± 0.49, $p < 0.05$) and Group 2 runners ($\overline{X}$ = 5.70 ng/ml ± 0.57) had significantly lower levels than controls ($p < 0.05$) (Figure 10).

Summary tables of means, standard deviations and standard errors for hormone levels in the 5, 4 and 3 group analysis appear in Appendix 7(a) and Appendix 7(b).

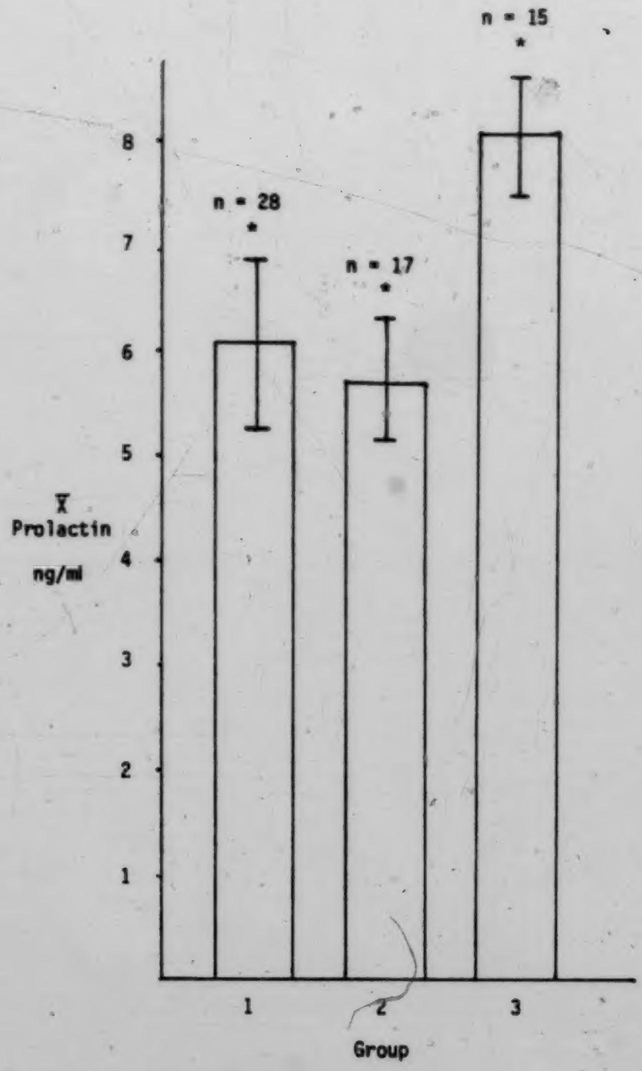
Percentage Differences in Hormone Levels

3 Group Analysis

Total, free and non-SHBG testosterone and prolactin as percentages of mean control leves are represented in Table 2(A, B and C).

Differences in total testosterone were not significant in either the 5, 4 or 3 group analysis although total T was in the 3 group analysis on average 15% lower in 40+ miles per week runners, 19% in 20-39 miles per week runners than controls. Free T was 35% lower in 40+ miles per week runners and 40% lower in 20-39 miles per week runners. Non-SHBG T was 26% lower in both 40+ miles per week runners and 20-39 miles per week runners. Prolactin was approximately 25% lower in both groups.

In the 4 group analysis similar findings were apparent when the 40+ miles per week group was divided into 70+ miles per week runners and 40-69 miles per week runners. In the 70+ miles per week group total T



* Group 1 vs 3 significantly different, $p < 0.05$

* Group 2 vs 3 significantly different, $p < 0.05$

Figure 10. Mean prolactin levels, 3 group analysis

TABLE 2

Percent Differences in Hormone Levels

3 Group Analysis

Table A

Percentage of Mean Control Levels

Group	Non-SHBG T	Free T	Total T	Prolactin
40+ miles per week	63.6	64.4	85.8	75.8
20-39 miles per week	64.2	59.9	81.1	75.2
Control	100	100	100	100

4 Group Analysis

Table B

Percentage of Mean Control Levels

Group	Non-SHBG T	Free T	Total T	Prolactin
70+ miles per week	66.6	61.9	80.0	85.0
40-69 miles per week	61.9	65.8	88.0	71.2
20-39 miles per week	64.2	59.9	81.1	76.2
Control	100	100	100	100

5 Group Analysis

Table C

Group	Non-SHBG T	Free T	Total T	Prolactin
	Percentage of Mean Control Levels			
University Runners	73.1	68.0	78.9	74.2
70+ miles per week	55.8	52.2	77.8	85.7
40-69 miles per week	55.8	61.8	87.7	71.8
20-39 miles per week	69.2	64.5	86.3	78.0
Control	100	100	100	100

was 20% lower, free T 38% lower and non-SHBG bound T 33% lower than in controls. Prolactin was on average 15% lower in the 70+ miles per week group than controls.

Total T was 12% lower, free T, 34% and non-SHBG bound T 38% lower than controls in the 40-69 miles per week group with prolactin being an average 29% lower than controls.

Total T was 19% lower, free T 40% lower and non-SHBG bound T 36% lower in the 20-39 miles per week group than controls.

Although differences in free and testosterone only became significant at the 3 and 4 group levels, a general picture of reduced free T is seen throughout the analysis progression. Differences in total testosterone, although not significant, are consistently around 20% lower in runners versus controls.

Low cell sizes and high variability in the 5 group analysis may be responsible for a lack of significance in differences between runners and controls. Non-SHBG bound testosterone was however significantly different at the 5 group analysis level.

Total T is seen to be 22% lower in the university and 70+ miles per week groups while only 13% lower in the 40-69 miles per week and 20-39 miles per week group. Free testosterone was between 38 and 32% lower the university, 40-69 and 20-39 miles per week groups but 48% lower in the 70+ miles per week runners. Non-SHBG bound T was 27% and 30% lower in in the university and 20-39 miles per week groups respectively but 45% in the 70+ and 40-69 miles per week groups. Prolactin was 26%, 14%, 28% and 22% lower in the university, 70+, 40-69 and 20-39 miles per week groups respectively.

Differences in total, free and non-SHBG bound testosterone and prolactin are thus apparent between all running groups and control groups.

Individual free T and non-SHBG bound testosterone levels and prolactin for the final 3 group analysis may be found in Figures 11, 12, 13 and 14.

PART III

Results

Spearman Rank Order Correlations (Part i)

Based on differences revealed by analysis of variance on 3 groups (40+, 20-39 miles per week and controls) and post hoc Scheffé tests selected comparisons were made between the following variables:

Waist Estimation Score - mean of measures
Mean Body Image Estimation - overall mean score
Eating Attitude Scores
Anxiety Scores
Self-Esteem Scores
Infrequency Scores
Total Testosterone
Free Testosterone
Non-SHBG Bound Testosterone
Prolactin
Luteinizing Hormone
Follicle Stimulating Hormone
Cortisol

The Spearman rank order correlation method was used given that numbers in groups were insufficient to give power to the use of a Pearson Product moment correlation. Results are reported in the Appendices in full and significant correlations are reported at the 0.05 and 0.01 levels of significance.

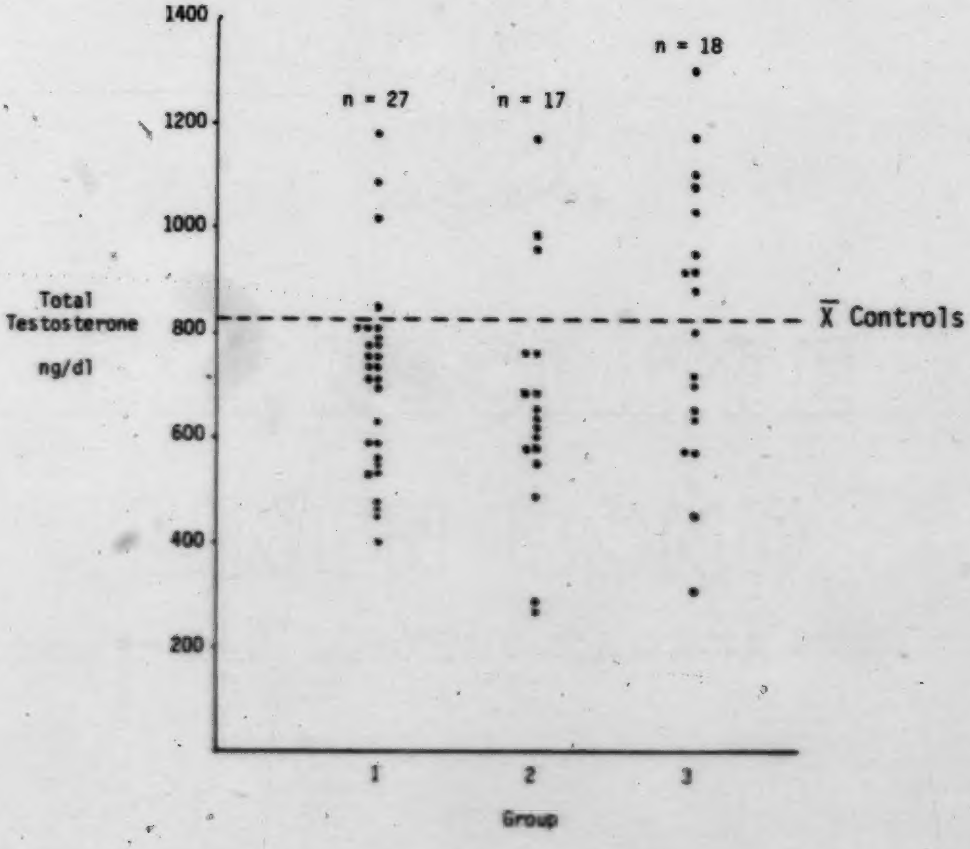


Figure 11. Individual total testosterone levels, 3 group analysis

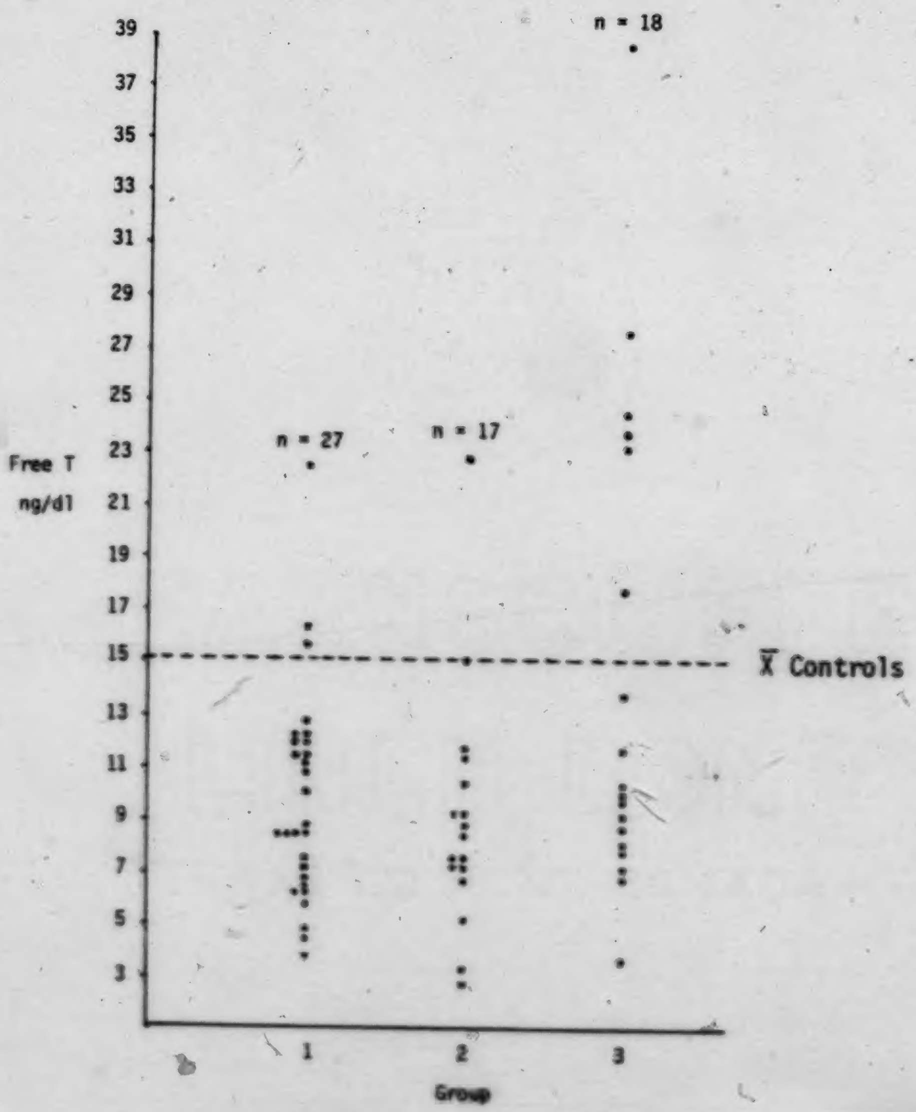


Figure 12. Individual free testosterone levels, 3 group analysis

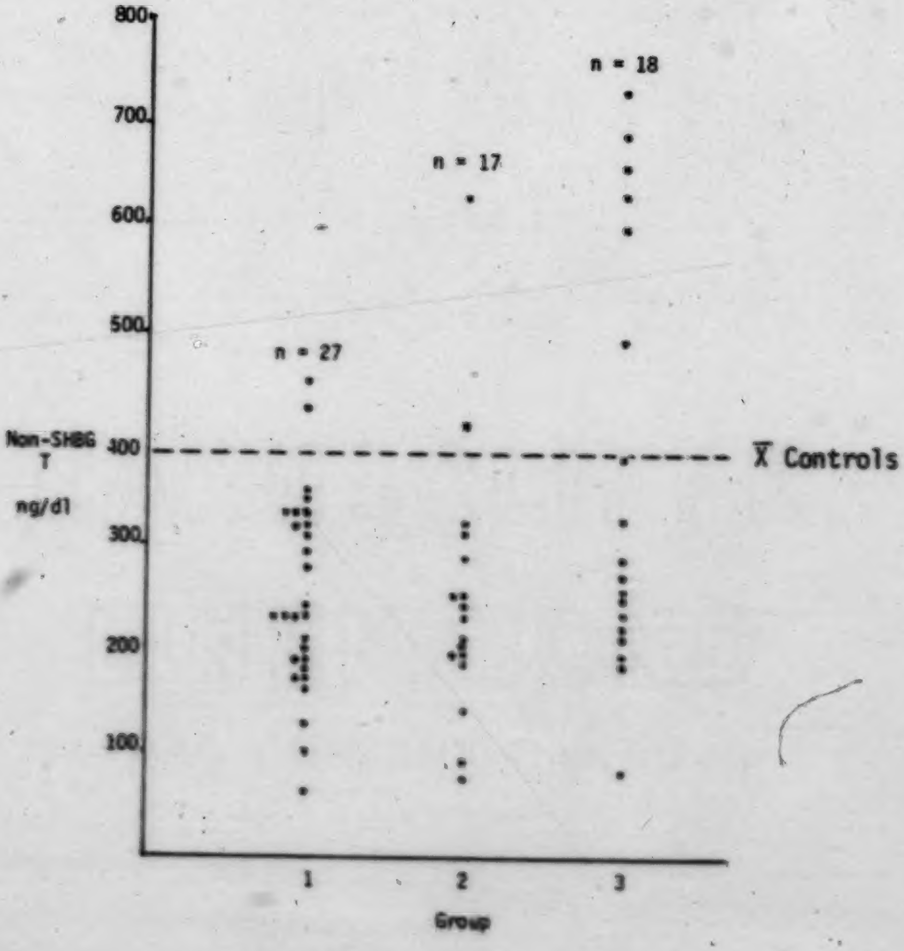


Figure 13. Individual levels - non-SHBG testosterone, 3 group analysis

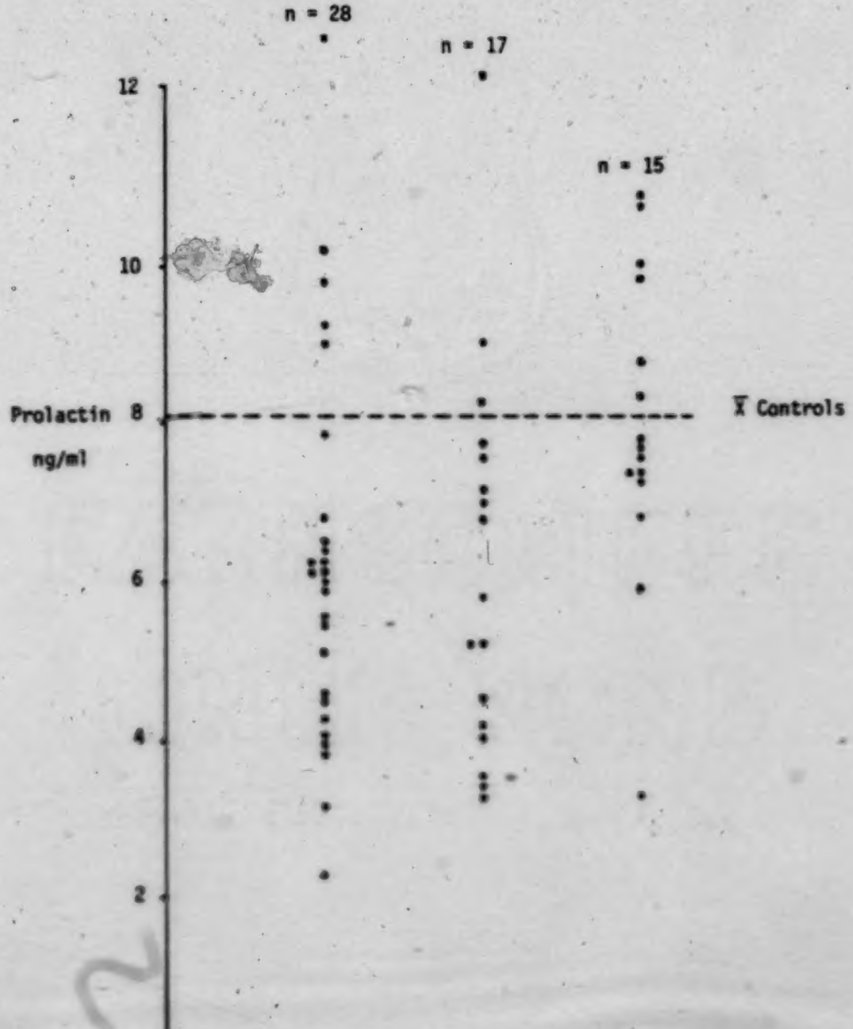


Figure 14. Individual prolactin levels, 3 group analysis

Correlations were computed for 3 separate groups, 40+ miles per week, 20-39 miles per week and control group. Again, this was based on ANOVA indications of major differences occurring among these groups. Significant correlations are reported below by group.

40+ Miles per Week - Spearman Rank Order Correlations

Waist width estimation scores correlated with mean body image estimation scores, $p = +0.937$ ($p < 0.01$), anxiety scores, $p = +0.410$ ($p < 0.05$), infrequency scores, $p = 0.419$ ($p < 0.05$) and prolactin levels $p = 0.844$ ($p < 0.01$).

Waist estimation correlated inversely with self-esteem levels, $p = -0.338$ ($p < 0.05$).

Mean body image overestimation correlated with infrequency scores, $p = 0.371$ ($p < 0.05$).

Eating attitude scores (EAT) correlated inversely with prolactin, $p = -0.331$ ($p < 0.05$), LH, $p = -0.427$ ($p < 0.05$) and cortisol $p = -0.635$, ($p < 0.01$).

Anxiety was inversely correlated to self-esteem $p = -0.396$ ($p < 0.05$) and to non-SHBG bound T, $p = -0.383$ ($p < 0.05$).

Infrequency scores correlated inversely to free testosterone (free T) scores, $p = -0.872$ ($p < 0.01$).

Total testosterone (total T) correlated significantly with free T, $p = 0.684$ ($p < 0.01$) and non-SHBG bound T, $p = +0.601$ ($p < 0.01$).

Free T correlated with non-SHBG bound testosterone (non-SHBG bound T), $p = 0.987$ ($p < 0.01$) and non-SHBG bound T correlated with prolactin levels, $p = +0.83$ ($p < 0.01$). Prolactin correlated with LH, $p = 0.511$ ($p < 0.01$)

and FSH, $p = 0.517$ ($p < 0.01$) and with cortisol, $p = 0.365$ ($p < 0.05$). LH levels correlated with FSH levels, $p = 0.376$ ($p < 0.05$). No other comparisons were significant.

20-39 Miles per Week Group - Spearman Rank Order Correlations

Waist width estimation scores correlated to mean body image estimation scores, $p = 0.747$ and prolactin, $p = 0.686$ ($p < 0.01$).

Inverse correlations were found between waist scores and total testosterone, $p = -0.910$, free T, $p = -0.778$, non-SHBG bound T, $p = -0.853$, FSH, $p = -0.613$, and cortisol levels, $p = -0.839$ ($p < 0.01$).

Mean body image estimation scores correlated inversely with total T, $p = -0.796$, free T, $p = -0.662$, non-SHBG bound T, $p = -0.566$, prolactin, $p = -0.491$, FSH, $p = -0.650$ and cortisol, $p = -0.608$ ($p < 0.01$).

EAT correlated with non-SHBG bound T, $p = -0.530$ ($p < 0.01$).

Self-esteem correlated inversely with free T, $p = -0.503$, and FSH, $p = -0.537$ ($p < 0.05$) and LH, $p = -0.658$ ($p < 0.01$).

Total T levels correlated with free T, $p = 0.920$, and non-SHBG bound T, $p = 0.968$ ($p < 0.01$) and with cortisol, $p = 0.431$ ($p < 0.05$).

Free T correlated with non-SHBG bound T, $p = 0.972$ ($p < 0.01$).

No other comparisons were significant.

Control Group - Spearman Rank Order Correlations

Waist width estimation scores correlated significantly with mean body image estimation scores, $p = 0.902$ and prolactin levels, $p = 0.796$ ($p < 0.01$) and with infrequency scores, $p = 0.462$ ($p < 0.05$).

Self-esteem correlated inversely to prolactin levels, $p = -0.705$ ($p < 0.01$).

Total T correlated with free T, $p = 0.921$, non-SHBG T, $p = 0.909$ ($p < 0.01$) and inversely with LH levels, $p = -0.480$ ($p < 0.05$).

Free testosterone correlated with non-SHBG bound T, $p = 0.971$ ($p < 0.01$) and inversely with LH levels, $p = -0.517$ ($p < 0.05$).

Non-SHBG T bound T correlated inversely with LH levels, $p = -0.468$ ($p < 0.05$).

No other comparisons were significant for the control group.

A matrices of correlations and significant correlations for the above variables may be found in Appendix 8(a) and Appendix 8(b).

Spearman Rank Order Correlations (Part ii)

Correlations of Ponderal Index and Frequency of Runs per Week with Other Variables

Spearman rank order correlations were computed for ponderal index (a measure of body linearity) and number of runs per week with mean waist estimation, mean body size estimation, EAT, anxiety, self-esteem, infrequency, total testosterone, free testosterone, non-SHBG bound testosterone, prolactin, FSH, LH and cortisol. Significant correlations were found between ponderal index (PI) and free T ($p = 0.320$, $p < 0.05$), in the 40+ miles per week group. In the 20-39 miles per week group pondural index correlated with cortisol ($p = 0.479$, $p < 0.05$) and inversely with self-esteem ($p = -0.4110$, $p < 0.05$). Total testosterone ($p = -0.397$, $p < 0.05$), non-SHBG bound T ($p = -0.390$, $p < 0.05$). A correlation of -0.360 was found between free T and pondural index. However, this was not statistically significant. In general, pondural index in the 20-39 miles per week group appeared to be related to the various measures of testosterone.

In the control group pondural index correlated inversely with total T ($p = -0.724$, $p \ll 0.05$) and free T ($p = -0.572$, $p < 0.05$). No other correlations between pondural index and the above-mentioned variables were significant.

The number of runs per week in the 40+ miles per week group correlated significantly with the EAT scores ($p = 0.549$, $p < 0.01$). There were no other significant correlations between number of runs per week and any of the other variables.

A matrix of correlations between the above-mentioned variables may be found in Appendix 9.

CHAPTER V

DISCUSSION

Demographic Data Questionnaire

Between group differences on hormone levels, body image perception or personality did not appear to be confounded by age or body stature (as indicated by ponderal index) as no significant differences were found between groups of runners and controls.

Demographic data from the present study revealed similar findings to those of Yates, Leehay and Shisslak (1983) in runners, and Bruch (1978) in anorexic patients. However, although habitual runners were found to come from white-collar, upper income bracket professions and high achievement orientated backgrounds as do many anorexic patients, it is entirely possible that such a group is self-selecting. Those in manual occupations might well not have the need or energy for prolonged fitness orientated activities such as running. The results of this study and of other studies revealing similar demographic characteristics in runners (Burdick and Zloty, 1973; Hartung and Farge, 1977; Gontang, Clitsome and Kostrubala, 1978; Clitsome and Kostrubala, 1978; Dowd and Innes, 1981) may therefore not be comparable to the demographic background of the anorexic patient. The runner from this type of background may not be motivated to run by the same pressures that contribute to the onset of Anorexia nervosa.

Running Commitment Questionnaire

Questions from the questionnaire may be divided into four categories for the purposes of the discussion: running and eating attitudes, running commitment, domestic and occupational responsibility and emotional attachment to running.

Eating attitudes as defined by the EAT revealed no significant differences between groups of runners and controls. Responses to the Commitment Questionnaire were consistent with this finding in that although 61% of the runners reported running at lunch time, only 26% frequently missed lunch in order to run. Only 4% admitted to missing a meal frequently. The apparent inconsistency in the latter responses will be discussed at a later point. Generally, responses to questions relating to meal avoidance were not consistent with anorexic-type behaviour. Similarly, the questionnaire revealed that 60% of the runners regularly consumed at least three meals per day which was not consistent with anorexic behaviour. These findings did agree with the generally low eating attitude scores through the running groups and therefore a hypothesis of anorexic eating attitudes in habitual runners was not supported.

Many anorexics feel a need to exercise directly after a meal (Bruch, 1971, 1978). However, only 20% of the runners in this study expressed an occasional need to exercise directly after eating. Once again, this finding was not indicative of hyperactivity associated with reduced caloric consumption.

Commitment to running was assessed via four questions. Forty-seven percent of the runners reported that running had a fundamental importance in their lives while only 14% said that running was primarily for enjoyment. It was clear from this and anecdotal evidence that running was clearly more than a means of enjoyment or acquisition of fitness. Previous reports on runners have suggested that negative addiction in the runner was finally characterized by a failure to adhere to medical advice (Morgan, 1979). Eighty-five percent of the runners utilized in the study said they would only partially adhere to medical advice to rest while 12% said they would totally ignore the advice. In response to a directive of total cessation of running for medical reasons, 15% said they would totally ignore this advice while 40% said they would experience an extreme sense of loss. These findings partially support the negative addiction construct suggested by Morgan (1979). Furthermore, conversation with the runners left little doubt as to the importance of running in their lives.

Emotions associated with running were elicited through questions regarding feelings on missing a daily run and the above mentioned question regarding medical advice to cease running. One-third of the runners said they would feel mild anxiety at missing the daily run while only one admitted to potential feelings of high anxiety. It did not appear that the runners demonstrated the addictive qualities alluded to by Morgan (1979) and Little (1981) on the basis of the questionnaire responses.

Responsibility of the runners was assessed according to responses regarding lateness for work and failure to fulfill domestic responsibilities. It has been reported that addicted runners frequently forego domestic and occupational responsibilities.(Morgan, 1979; Yates et al., 1983). It is also well documented that the bizarre behaviour of the anorexic interferes with familial and occupational responsibility (Bruch, 1973, 1978). Only 6% of the runners in the present study reported that running often made them late for work and only 8% said that running caused them to neglect domestic responsibility. Running did not appear to interfere with domestic and occupational responsibilities of the runners.

Body Image

The Body Image Perception test revealed a general phenomenon of overestimation of body widths in all running groups and controls. However, there was a particular trend towards increasing overestimation of waist width with increasing mileage in the runners. A large variation in width perception among the runners and small group numbers in all likelihood accounted for the lack of significance of differences when results were analyzed using four and five groups. However, when the runners were divided into a high mileage group of 40+ miles per week and a low mileage group of 20-39 miles per week, the high mileage group demonstrated a significantly larger overestimation of waist width than the low mileage runners or controls. The general overestimation of body width in all subjects is consistent with previous studies comparing anorexics and normals (Casper, Halmi et al., 1979; Halmi et al., 1977). As anorexics are prone to overestimate bodily dimensions compared to normals (Casper et al., 1979; Halmi et al., 1977), then so the runners in the present study appeared to overestimate body size compared to controls. The runners as a group were generally much slimmer than controls and yet they overestimated body size more than controls. Again, this is consistent with the anorexic tendency of overestimation with increasing emaciation. Unfortunately, however, this test could not be carried out on anorexics for ethical reasons. Comparisons to anorexic body image perception must therefore be speculative.

If the general overestimation with waist size was associated to a concern with weight, the results might be compared to Askevold's (1975) notion of locii of concern with certain bodily dimensions according to various ailments. Anorexic patients were found to specifically overestimate waist size which, according to Askevold, was indicative of their desire for thinness. It may be that as a runner increases mileage and perhaps loses weight then so his concern with body size grows. This remains purely speculative and a longitudinal study would be required to assess the effect of increasing mileage on body image perception.

Eating Attitude Scores

Analysis of eating attitude scores revealed no significant differences among groups at any analysis level. Group mean scores were consistent with the normal mean score of 10 as defined by Garner and Garfinkel (1979). Results did not suggest the presence of abnormal eating attitudes in runners as found in female runners by Henry (1982), in ballet dancers (Garner and Garfinkel, 1979) or in anorexic patients (Bruch, 1971, 1973, 1978). A hypothesis of similar eating attitudes in runners and anorexics was not supported.

Jackson Personality Inventory

The Jackson Personality Inventory did not reveal a typical profile of runners according to mileage or as a group compared to controls. High anxiety in long distance runners found in previous studies (Morgan, 1968, 1978) or low anxiety in distance runners (Costill and Morgan, 1976; Morgan, 1974) was not identified by the JPI in the present group nor were the runners less socially adroit or reserved than normals as found in previous studies (Hartung and Farge, 1977; Burdick and Zloty, 1973). Low self-esteem (Bruch, 1978), high anxiety and low social adroitness (Halmi, 1974) or excessive shyness (Kay and Leigh, 1956) traits attributed to the anorexic patient in previous studies were not evident from the results of this study. However, when the analysis was conducted at the four and three group levels, significantly higher infrequency scores were found in the high mileage running groups versus controls. High infrequency scores are interpreted as a measure of improbably or inconsistent responses and as such this factor will be discussed further.

Results of the questionnaires did not point to clear comparable characteristics between runners and anorexics. However, some disparity between anecdotal evidence was gained from the study and written questionnaire responses. Such a disparity may possibly be an expression of denial as found in anorexic patients. The significantly higher infrequency scores on the JPI may similarly reflect this denial.

In summary, the questionnaires failed to identify the addiction or commitment to running and concerns with body size and eating as described by previous authors (Yates et al., 1983; Morgan, 1979; Little, 1981). Three explanations are thus possible for the disparities between anecdotal and written responses.

Firstly, the questionnaires used were either inappropriate or lacked the sensitivity to identify the above mentioned characteristics. Secondly, it may be that the questionnaires were valid tools for the measurement of eating attitudes and commitment to running but that runners were not concerned with body weight, caloric content of food, or addicted to running as suggested by Yates, Leehay and Shisslak (1983) or Morgan (1979) or Little (1981). Anecdotal evidence suggesting the opposite of this may have applied to a few specific cases. Thirdly, habitual runners may be negatively addicted to running, may be concerned with body weight and food consumption, but deny these characteristics in written questionnaires as the anorexic denies illness. The latter may be supported, as previously suggested by significantly higher infrequency scores on the JPI in the high mileage running group compared to controls.

The table of similarities between habitual running and AN may thus be revised in the light of results from the present study (see Table 3).

Several studies have examined the acute effects of endurance running on total testosterone levels prior to competition and stressful endurance running (Morville et al., 1979; Kuoppasalmi, 1980; Dessypris et al., 1976). However, no attention has been paid to resting levels of total, free or non-SHBG bound testosterone in endurance runners. The present study therefore involved basal sampling after a 24 hour rest period and the absence of recent competition.

Studies that have documented pre-race testosterone levels reveal values with the normal 300-900 ng/dl range, for adult males. Dessypris, Kuoppasalmi and Adlercreutz (1976) reported levels of 662.08 ng/dl prior to a marathon run, Kuoppasalmi (1980), 504 ng/dl in runners during a normal day and 725.62 ng/dl prior to an endurance run. Schmid et al. (1982) reported levels of 550.08 ng/dl in a group of skiiers prior to a 36 km cross country ski race. Such values are used as control samples, yet do not take into account any anticipatory change in testosterone prior to exercise (Cumming and Rebar, 1983). Morville et al. (1979) reported levels of testosterone of 376 ng/dl in runners and 502 ng/dl in controls but did not statistically evaluate nor discuss this difference. The reduced testosterone levels induced by acute bouts of exercise have also been found to recover to above pre-exercise resting levels (Morville et al., 1979; Kuoppasalmi, 1980).

Testosterone levels of the runners used in the present study were within the normal range for males except for 2 subjects outside the lower range. Mean levels of 708 ng/dl were found in the 40 plus miles

Table 3

SIMILARITIES BETWEEN HABITUAL RUNNING AND ANOREXIA NERVOSA: REVISED ACCORDING TO FINDINGS OF THE STUDY

Habitual Running	Anorexia Nervosa
1. Overestimation of waist size compared to normals.	1. Consistent overestimation of body size.
2. Weight control not of prime importance to the runner.	2. A relentless pursuit of thinness and pre-occupation with food and weight.
3. No concern with caloric content of food.	3. Pre-occupation with food and caloric content of food.
4. No apparent anxiety related to cessation of running.	4. Denial of illness.
5. Normal eating behaviours and self-esteem levels. No indication of feelings of loss of self-control.	5. Food avoidance: a means of achieving self-control.
6. Rituals of running.	6. Rituals of self-imposed starvation.
7. Hyperactive characteristics of habitual runners. Activity important to feelings of well being rather than to burn off unwanted calories.	7. Hyperactivity in AN patients. Goalless activity to burn off calories.
8. Endocrine profile of the male habitual runner does not resemble that of the AN patient. T levels, LH, FSH, cortisol, T_4, TSH within the normal range. Prolactin levels reduced.	8. Endocrine disorders in male and female AN patients. Reduced T in the male, increased T in the female. Reduced LH, FSH in male and female AN. Increased prolactin, growth hormone and cortisol levels in both sexes.

per week group and 669 ng/dl in the 20-39 miles per week group. This compared to mean levels of 802 ng/dl in controls.

Differences among the 40 plus miles per week, 20-39 miles per week and control groups were most marked when levels of free T and non-SHBG bound T were calculated. Total testosterone was approximately 15% lower in the running groups than controls, whereas free T was between 35-40% lower and non-SHBG bound T, 26% lower in runners (40+ and 20-39 miles per week) than controls. The differences were significant at the 0.05 level for free T and non-SHBG bound T. Possible mechanisms and implications of reduced testosterone levels may be examined.

Testosterone levels in the blood depend on three factors: synthesis, binding and clearance. Reduced testosterone levels may therefore reflect reduced testicular synthesis, alterations in the binding of testosterone to specific binding globulins or an increase in the metabolic clearance rate.

Clearance of testosterone is hepatic and extrahepatic. Muscles contribute to metabolic clearance and possess both specific androgen receptors (Snochowski et al., 1981). and enzymes capable of metabolizing testosterone (Stenstead and Eik-Ness, 1981). Reduced testosterone levels have been found in rats after endurance training accompanied by increased excretion of urinary by-products of steroid metabolism (Dohm, 1978). The possibility of increased metabolic clearance of testosterone via increased intramuscular utilization is impossible to determine in that there is no known specific metabolite of testosterone from muscle clearance.

Production of testosterone is dependent on LH released in a pulsatile fashion from the anterior-pituitary. Prolactin may also play a role in sensitizing the Leydig Cells of the testes fo LH, thereby facilitating or enhancing the secretion of testosterone into the circulation. The production rate (PR) of testosterone is dependent on the serum levels (SL) and metabolic clearance rate (MCR) of the hormone expressed as,

$$PR = SL \times MCR$$

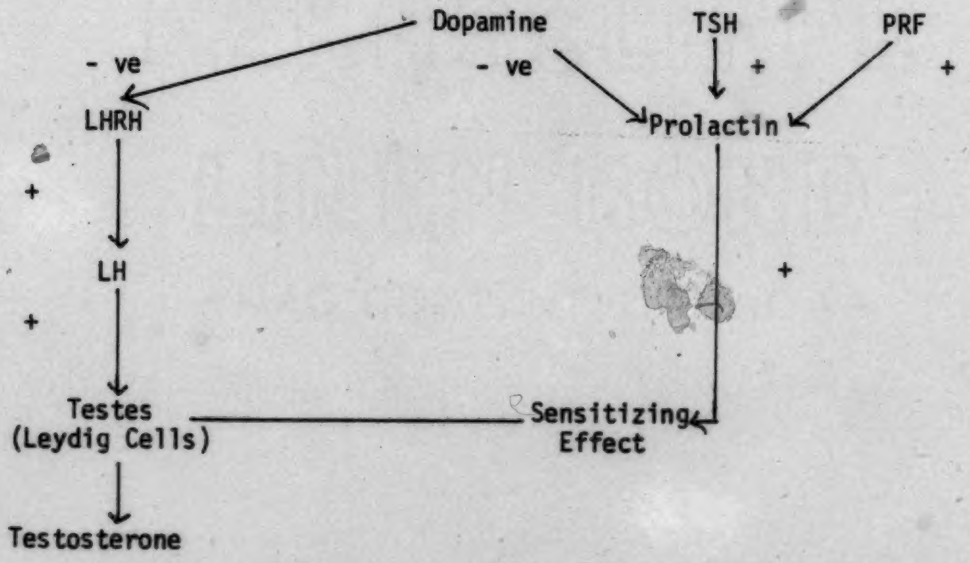
Assessment of the metabolic clearance rate is, however, complex and thus its contribution to reduced T levels difficult to ascertain. However, non-SHBG bound T and MCR are significantly correlated and a linear relationship also exists between free T and Dihydrotestosterone (DHT) and MCR (Vermeulen, 1969). The present findings suggest, therefore, a reduction rather than increase in the metabolic clearance rate of testosterone. Factors that may have affected testosterone production may be examined.

Reduced prolactin levels have been associated with reduced testicular production of testosterone (Besser and Thorner, 1976). Prolactin has also been found to act as a sensitizing agent on the Leydig cells of the testes to LH (Bartke and Dalterio, 1976; Hafiez, Lloyd and Bartke, 1972). Significantly reduced prolactin levels in the runners (40+ miles per week and 20-39 miles per week) may be associated with reduced testosterone levels in the runners.

LH levels were found to be in the normal range for adult males (3.6 to 22.4 mIu/ml). As such reduced LH levels would not appear to be responsible for reduced testosterone levels. However, only one sample was taken and this may not account for the pulsatile release of LH in males (Naftolin, Judd and Yen, 1973). Furthermore, it has been found in adolescents that there is a marked discrepency between LH measured by RIA and that measured by Bioassay (Lucky et al., 1980). A single sample of LH is therefore not conclusive evidence of either an impaired or normal release of LH.

Acute elevation of circulating cortisol levels have been shown to decrease testosterone levels, apparently by a direct action on the testes (Doerr and Pirke, 1976; Cumming, Quigley and Yen, 1983). In studies of long distance runners acute hypercortisolemia has also been associated with reduced testosterone levels (Morville et al., 1979). Cortisol levels were within the normal range for runners (3.0-11.0 ug/dl). No evidence was found to support a hypothesis of cortisol induced suppression of circulating testosterone levels.

Dopamine may have an effect on the release of the LH precursor, luteinizing hormone releasing hormone (LHRH) and prolactin. This is explained in the following diagram.



It is possible that an increased dopaminergic drive may have reduced the pulsatile release of LHRH and LH and prolactin. Although dopamine regulates both LHRH and prolactin it is likely that the regulatory systems are separate. This area warrants further investigation.

Age differences did not appear to be responsible for reduced testosterone levels in the runners. No significant correlations between age, total, free and non-SHBG bound T were found. This is in agreement with the finding of Vermeulen et al. (1971, 1972) who found large variations in testosterone levels across comparable age groups.

The affects of diet on testosterone levels may be examined. A high fibre, low saturated fat diet has been associated with significantly reduced total and free testosterone levels (Hamalainen et al., 1983). Although there was no accurate caloric assessment during the present study, eating attitudes and the number of meals eaten per day appeared normal in the majority of runners. If the above-mentioned measures are indicative of normal dietary behaviour then diet does not appear to be a factor in the reduced testosterone levels. However, no conclusive statement can be made until further investigation involving accurate caloric assessment over a period of time has been conducted.

At this point the implications of reduced total, free and non-SHBG bound testosterone should be considered.

Since men do not possess a critical dependence upon a regular cyclic functional pattern of the endocrine system, it would seem possible that sperm production would be unaffected by minor changes in reproductive hormone levels. However, reduced testosterone levels may in part be responsible for the reduced libido and potency anecdotally expressed in some runners as reduced testosterone levels are suggested as a cause of reduced potency and libido in anorexic males (Beumont, 1972).

It has been found that testosterone decreases muscle degradation brought about by endurance exercise in rats (Dahlmann et al., 1981). If testosterone secretion is lower in runners then this might have implications for reduced protein synthesis in the muscle. Furthermore, it has been found that both free and non-SHBG bound testosterone can freely enter extra-hepatic cells for clearance although the fraction available for extra-hepatic clearance is unclear (Rivarola, Singleton, Migeon, 1967; Baird et al., 1969; Pardridge, 1981). If free and non-SHBG bound testosterone represent the bioavailable fraction of testosterone then reduced levels in the runners might have considerable implications for tissue repair and performance.

Whereas anorexics present with reduced gonadotropic hormone levels (Beumont et al., 1978; Brown et al., 1977) there were no significant differences between running groups and controls and LH and FSH levels. Nor were LH or FSH levels in the anorexic range. The impaired pituitary-thyroid function of the anorexic as demonstrated by reduced T_3 levels and increased rT_3 (Burman et al., 1977) was not evident from the results of the present study. Pituitary-adrenal function is altered in anorexic patients associated with raised cortisol levels (Alvarez, 1972; Brown et al., 1977; Vigersky and Loriaux, 1977). The runners in the present study presented no evidence of raised cortisol levels. Reduced prolactin levels found in the runners were not consistent with raised prolactin levels in anorexic patients (Garfinkel and Garner, 1982). Testosterone, though, reduced in runners were not in the range usually reported in anorexic males (Beumont et al., 1970; Beumont, 1972). The majority of the runners had levels within the normal 300-900 ng/dl range for adult males.

The reduced total testosterone and significantly reduced free and non-SHBG bound testosterone levels in the runners is at this point unexplained. Further investigatory measures may thus be proposed.

Firstly, the pulsatile release of LH should be examined in the runners in order to ascertain the presence or absence of a pituitary malfunction. Secondly, provocative tests using LHRH might be implemented in order to assess the pulsatile response of LH to LHRH pulses. Thirdly, dopamine antagonists might be utilized in order to investigate a possible normalizing effect on testosterone in the runners. Finally, an animal model might be used in order to assess the effects of prolactin and dopamine antagonists on testosterone production.

Correlations of Ponderal Index and Number of Runs per Week with Other Variables

Ponderal index (PI: a measure of linearity) was significantly correlated to free T ($p < 0.05$) in the 40 plus miles per week group. This suggested that a high degree of linearity in these runners was correlated to the highest testosterone levels. However, PI was inversely correlated to free total, and non-SHBG bound T ($p < 0.05$) in the 20-39 miles per week runners. Linearity seemed therefore to be related to low T levels in this group. It should also be considered that this group had the lowest total, free and non-SHBG bound T levels of the two running groups and indeed of all groups at the 4 group analysis level. PI in 20-39 miles per week runners was inversely correlated to self-esteem ($p < 0.05$) suggesting that low self-esteem was related to linearity. However, PI was also inversely correlated to total and free testosterone ($p < 0.05$) in the control group. This may suggest a relationship of testosterone levels to body weight rather than to a mileage factor. PI was also inversely related to cortisol levels ($p < 0.05$) in the 20-39 miles per week group. The link between PI, cortisol and testosterone levels is, however, tenuous in that cortisol and testosterone levels were positively correlated in the 20-39 miles per week group. If PI, cortisol and T levels were related an inverse relationship between cortisol and testosterone might have been expected. However, it should be noted that waist estimation scores were inversely related to total, free and non-SHBG bound T in the 20-39 miles per week group. This may suggest that linear runners as indicated by PI, who overestimate body size, also have lower, total, free and non-SHBG testosterone levels.

The number of runs per week were also correlated with the other measured variables and it was found that EAT scores in the 40 miles per week runners were positively and significantly correlated to the number of runs per week. Examination of the data reveals, for example, that one runner scored 19 on the EAT and ran 13 times per week and that another ran 14 times weekly, scoring 24 on the EAT. However, scores for the EAT are generally low and thus this finding is not indicative of a relationship of anorexic eating attitudes to the number of runs per week.

Variable by Variable Correlations

A summary table of correlations between variables may be found in Appendix 8(a).

Waist scores correlated +0.937 ($p < 0.01$) with mean body image estimation, +0.747 ($p < 0.01$) and +0.902 ($p < 0.01$) in the 40+ miles per week runners, 20-39 miles per week runners and controls respectively by Spearman rank order correlation. Waist estimation correlated with anxiety ($p < 0.05$) in the 40+ miles per week runners, although not in the other groups. A relationship between overestimation of waist size and anxiety level was apparent in relation to mileage. The waist score was also inversely related to self-esteem in the 40+ miles per week group ($p < 0.05$) suggesting low self-esteem in relation to overestimation of waist width. Anxiety and self-esteem were correspondingly inversely related ($p < 0.05$) in this group. High waist scores were also positively correlated with high infrequency scores in the 40+ miles per week group ($p < 0.05$). However, the infrequency scores in this group were not related to anxiety or self-esteem scores. The mean body image perception score was also correlated to infrequency scores ($p < 0.05$) suggesting a relationship of general overestimation of body image perception and infrequency as measured by the JPI.

The foregoing discussion thus demonstrates an apparent relationship of personality characteristics to body image estimation in the high mileage runners though not in the 20-39 miles per week group. On correlating body image, EAT and personality scores to hormone levels a different picture appears in that most significant hormone and psychological relationships appear in the 20-39 miles per week group.

Eating attitudes correlated inversely with prolactin, LH ($p < 0.05$) and cortisol ($p < 0.01$) in the 40+ miles per week group. These relationships did not appear in the low mileage or control groups. Although EAT scores are also positively correlated to the number of runs per week in this group, the EAT scores are too low to attach any significance to this finding.

Waist measures were positively correlated to prolactin in the 40+ miles per week group ($p < 0.01$). However, prolactin levels do not correlate with testosterone levels either positively or inversely in this or the lower mileage group. An association of prolactin levels, testosterone levels, PI and body image is therefore not apparent.

High anxiety scores were significantly and inversely correlated to non-SHBG bound T in the 40+ miles per week ($p < 0.05$), although not in either of the other groups. However, high anxiety levels and low free and total testosterone levels are not related in the high mileage group or low mileage runners. Anxiety levels and T levels would not appear to be highly related.

Self-esteem did not correlate significantly with any hormone levels in the 40+ miles per week runners although it correlated inversely with free T, LH and FSH in the 20-39 miles per week runners ($p < 0.05$). However, LH and T levels were not correlated in this group. A positive correlation between self-esteem and free T might have been expected if poor self-esteem was truly related to runners with low T levels, a high PI score and waist estimation score.

Infrequency scores were inversely correlated to free T levels in the 40+ miles per week runners ($p < 0.01$) although not with total or non-SHBG bound T. This relationship is not present in the other groups. In the high mileage group, there did seem to be a relationship between high waist scores, high anxiety, low self-esteem, high infrequency scores and a low free T fraction.

It is also interesting to observe that although T levels and LH are related in the control group ($p < 0.05$), there was no consistent relationship in the running groups. This may suggest hormonal imbalances in these groups.

In summary, various interrelationships among the measured variables do point to endocrine and psychological relationships. However, the various correlations tend to be inconsistent in their appearance within the groups.

Conclusions

The objective of the foregoing study was to compare eating attitudes, body image perception, personality and endocrine status between habitual runners and non-exercising controls and to investigate speculations of anorexic behaviours and tendencies in habitual runners.

Runners shared similar demographic characteristics to anorexic patients. Body image perception as defined by the image marking method of Askevold (1975) appeared to be most distorted in the high mileage running group (40+ miles per week). Waist size estimation was particularly overestimated in runners although the present findings agreed with previous findings of body size overestimation being present in normal individuals.

Runners did not, however, display similar eating attitudes, as defined by the EAT, as anorexics nor did they share similar personality traits or endocrine disorders as found in anorexic patients.

The phenomenon of denial of illness is common in anorexic men and women and the significantly higher infrequency scores on the JPI of the high mileage runners in conjunction with below normal scores on the EAT may be indicative of a denial of concern with caloric values of food in the runners. Anecdotal evidence collected during the study indicated a far greater concern with eating and body weight than was evident from questionnaire responses. The author thus concludes that the questionnaire material used might not be sensitive enough to the true concerns of the runners. On the other hand, if denial of concern with weight or caloric value of food is present in the runners then any direct type of questionnaire would most likely draw similar responses to those found.

Running commitment as measured by the Running Commitment Questionnaire was not as evident as previously expressed by other authors. The findings of significantly reduced free and non-SHBG bound testosterone in the runners as a group may be related to a chronic effect of prolonged participation in endurance running. This may have implications with regard to sexual potency, libido and protein synthesis during tissue repair in high mileage runners. The study provides for the first time evidence of possible chronic effects of endurance running on testicular secretion or metabolic clearance of testosterone.

Although the study did not provide conclusive evidence of true anorexic tendencies in runners, some of the findings do suggest the existence of similar behavioural and psychological traits in runners, as found in anorexics and therefore supports the speculation of previous authors in the area of study.

Recommendations for Further Research

From an essentially investigative study there have arisen grounds for further research into behavioural, psychological and endocrine characteristics of runners as compared to the true anorexic male. However, for an accurate comparison to be made a study must be conducted involving a trained behavioural psychologist and endocrinologist and true Anorexia nervosa patients. Furthermore, comparative aural and written responses to questionnaires related to eating attitudes and concern with weight should be collected to establish the presence or absence of a denial of concern with weight and food consumption and/or obsession with running similar to the denial of illness found in the anorexic.

To establish the existence of chronic affects of endurance running on testosterone production a further study should be conducted utilizing a larger sample of habitual runners and utilizing repeated measures of testicular androgens.

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APPENDICES

APPENDIX 1

Consent Form #1

Description of the study

The study proposal comprises:

(i) Completion of a running commitment questionnaire
(ii) Completion of an Eating Attitude Inventory
(iii) Completion of the 16 Factor Jackson Personality inventory
(iv) Participation in an assessment of body image using an image marking method
(v) The taking of a blood sample for the purposes of a plasma hormone analysis
(vi) Completion of a Demographic Data Questionnaire

Commitment

Subjects will be asked to fill out the questionnaires, take a body image test and be subjected to a blood sampling standard procedure. The time commitment will be approximately 2 hours.

I ______________________________ agree to take part in the above study and understand that my commitment to the study involves the answering of questionnaires relating to personality and the psychological profile on eating attitude inventory designed to elicit attitudinal responses to food and to eating, and a questionnaire designed to assess running commitment. I also agree to taking part in a simple test designed to assess the body image. I agree to the taking of a 20 ml blood sample.

I am aware that I may withdraw from the study at any time should I wish to do so. I also understand that the administration of the questionnaires will be confidential and by I.D. number and that the results of the questionnaires and body image test will be treated in the same manner.

Signed ________________ Subject

________________ Researcher

Date ________________

The information gained from the following set of questionnaires drawing test and blood sample will be analyzed by subject identity number. Subjects are assured that all the information will be treated with the utmost confidentiality and any resulting information will be available for inspection at any time that is required.

1. ANSWER THE QUESTIONNAIRES IN THE ORDER THAT THEY ARE PRESENTED IN THE FILE IN FRONT OF YOU.
2. ANSWER ALL QUESTIONS. PLEASE CHECK THAT YOU HAVE DONE SO WHEN YOU FINISH.
3. PLEASE ANSWER THE QUESTIONS HONESTLY.
4. THE SUCCESS OF THIS STUDY DEPENDS ON YOUR FOLLOWING THE INSTRUCTIONS PROVIDED TO THE LETTER.
5. ENSURE THAT YOUR IDENTITY NUMBER IS ON EVERY QUESTIONNAIRE.

MANY THANKS FOR YOUR TIME AND EFFORT.

G.D.W.

DEMOGRAPHIC DATA

QUESTIONNAIRE

(circle appropriate response)

(1) I.D. #:

(2) AGE:

(3) HEIGHT (cm):

(4) WEIGHT (kg):

(5) What is your present profession: ____________________________

(6) Income Bracket:

(a) 30,000 +
(b) 25,000 - 30,000
(c) 20,000 - 25,000
(d) 15,000 - 20,000
(e) below 15,000

(7) Education:

(a) High School
(b) Bachelor Degree
(c) Masters
(d) Ph.D. - M.D.
(e) other

(8) Did your parents encourage you to achieve in school:

(a) yes
(b) no

(9) At what age did you start running: _________________________

(10) What is the key motivational factor that resulted in your regular running program: (i.e., get in shape, hobby, enjoyment, lose weight)

____

____

RUNNING QUESTIONNAIRE

Answer all the following questions carefully. Accuracy of answers is essential to the success of the study. Where choices are given, circle (a, b, c, etc.) the most appropriate answer or category.

1. How far do you run each week?

   a. 20 - 40 miles per week
   b. 40 - 70 miles per week
   c. 70+ miles per week

2. How many times a week do you run? ____________________

3. Do you compete in races?

   a. Never
   b. Occasionally
   c. Frequently
   d. Every opportunity

4. During which period of the day do you usually run?

   a. Early morning
   b. Between breakfast and lunch
   c. Between lunch and supper
   d. After supper
   e. More than once (circle the periods)

5. Do you ever miss a meal in order to run?

   a. Miss breakfast
   b. Miss lunch
   c. Miss supper
   d. Never miss a meal
   e. Occasionally miss a meal
   f. Regularly miss more than one meal

6. Has running ever made you late for work?

   a. Never
   b. Occasionally
   c. Often

7. How many meals do you eat per day? (not snacks)

   a. 1
   b. 2
   c. 3
   d. more than three

8. Has running ever made you put off domestic responsibilities?

   a. Never
   b. Occasionally
   c. Often

9. What significance does running have in your life? (Circle/check more than one if applicable. Give priority, i.e., 1, 2, 3 to your responses.)

   a. A hobby
   b. A means of enjoyment
   c. A major part of your life which you feel you would not wish to give up
   d. A means of losing weight
   e. To become fit

10. Assume that you have a mild leg injury. Your doctor says do not run for three weeks. Would you . . .

    a. Obey him to the letter
    b. Rest a week and try the leg out
    c. Ignore him and run anyway

11. How do you feel if you miss a regular or daily run?

    a. Indifferent/neutral
    b. Mildly regretful
    c. Anxious
    d. Extremely anxious and restless

12. Do you ever feel the need to run directly after a meal?

    a. Always
    b. Sometimes
    c. Never

13. Assume you are told that you must give up running for good. Write down the first thought that comes into your head . . .

EATING ATTITUDES TEST

Please place an (X) under the column which applies best to each of the numbered statements. All of the results will be strictly confidential. Most of the questions directly relate to food or eating, although other types of questions have been included. Please answer each question carefully. Thank you.

	Always	Very Often	Often	Sometimes	Rarely	Never
1. Like eating with other people	( )	( )	( )	( )	( )	( )
2. Prepare food for others but do not eat what I cook	( )	( )	( )	( )	( )	( )
3. Become anxious prior to eating	( )	( )	( )	( )	( )	( )
4. Am terrified about being overweight	( )	( )	( )	( )	( )	( )
5. Avoid eating when I am hungry	( )	( )	( )	( )	( )	( )
6. Find myself preoccupied with food	( )	( )	( )	( )	( )	( )
7. Have gone on eating binges where I feel that I may not be able to stop	( )	( )	( )	( )	( )	( )
8. Cut my food into small pieces	( )	( )	( )	( )	( )	( )
9. Aware of the calorie content of food that I eat	( )	( )	( )	( )	( )	( )
10. Particularly avoid foods with a high carbohydrate content (e.g., bread, potatoes, rice, etc.)	( )	( )	( )	( )	( )	( )
11. Feel bloated after meals	( )	( )	( )	( )	( )	( )
12. Feel that others would prefer if I ate more	( )	( )	( )	( )	( )	( )
13. Vomit after I have eaten	( )	( )	( )	( )	( )	( )
14. Feel extremely guilty after eating	( )	( )	( )	( )	( )	( )
15. Am preoccupied with a desire to be thinner	( )	( )	( )	( )	( )	( )
16. Exercise strenuously to burn off calories	( )	( )	( )	( )	( )	( )
17. Weigh myself several times a day	( )	( )	( )	( )	( )	( )

		Always	Very often	Often	Sometimes	Rarely	Never
18.	Like my clothes to fit tightly	( )	( )	( )	( )	( )	( )
19.	Enjoy eating meat	( )	( )	( )	( )	( )	( )
20.	Wake up early in the morning	( )	( )	( )	( )	( )	( )
21.	Eat the same foods day after day	( )	( )	( )	( )	( )	( )
22.	Think about burning up calories when I exercise	( )	( )	( )	( )	( )	( )
23.	Other people think that I am too thin	( )	( )	( )	( )	( )	( )
24.	Am preoccupied with the thought of having fat on my body	( )	( )	( )	( )	( )	( )
25.	Take longer than others to eat my meals	( )	( )	( )	( )	( )	( )
26.	Enjoy eating at restaurants	( )	( )	( )	( )	( )	( )
27.	Take laxatives	( )	( )	( )	( )	( )	( )
28.	Avoid foods with sugar in them	( )	( )	( )	( )	( )	( )
29.	Eat diet foods	( )	( )	( )	( )	( )	( )
30.	Feel that food controls my life	( )	( )	( )	( )	( )	( )
31.	Display self control around food	( )	( )	( )	( )	( )	( )
32.	Feel that others pressure me to eat	( )	( )	( )	( )	( )	( )
33.	Give too much time and thought to food	( )	( )	( )	( )	( )	( )
34.	Suffer from constipation	( )	( )	( )	( )	( )	( )
35.	Feel uncomfortable after eating sweets	( )	( )	( )	( )	( )	( )
36.	Engage in dieting behaviour	( )	( )	( )	( )	( )	( )
37.	Like my stomach to be empty	( )	( )	( )	( )	( )	( )
38.	Enjoy trying new rich foods	( )	( )	( )	( )	( )	( )
39.	Have the impulse to vomit after meals	( )	( )	( )	( )	( )	( )

APPENDIX 2(a)

DEMOGRAPHIC DATA

Table 1. Age Group of Runners

Group	N	Age Range	$\bar{X}$ Age
University	10	18-25	21.8
70+	6	27-56	38.8
40-70	15	18-50	36.3
20-40	18	25-49	39.7
Controls	18	18-42	27.68

Table 2. Height and Weight of Runners

N	Group	Height Range (cm)	Mean Height (cm)	Weight Range (kg)	Mean Weight (kg)	Ponderal Index
10	Univ.	171.4 - 186.7	179.53	63.18 - 76.36	69.44	13.08
6	70+	160.5 - 177.8	171.75	59.09 - 73.63	65.07	12.84
15	40-70	161.7 - 187.9	178.57	60.90 - 78.18	68.19	13.08
18	20-40	170.2 - 182.9	176.54	61.36 - 90.90	67.43	12.72
18	Controls	171.5 - 193.0	180.44	63.63 - 97.72	73.98	12.92

Table 3. Occupation Categories of Runners and Controls

Group	Professor Teacher	Business Management	Medical Physician	Lab Tech.	Legal (Lawyers)	Govern- ment	Editor Journalism	Civil Engineering	Student	Other Non-White Collar Occupations
University (n = 10)	-	-	-	-	-	-	-	-	10	-
70+ (n = 6)	3	2	-	-	-	-	-	1	-	-
40-70 (n = 15)	2	2	1	3	2	1	1	1	2	1
20-40 (n = 18)	3	9	-	1	1	1	1	-	1	1
Controls (n = 18)	1	-	-	-	-	-	-	-	17	-
Total Runners	8	12	1	4	3	2	2	1	13	2
Total Controls	1	-	-	-	-	-	-	-	17	2

Table 4. Income Bracket of Runners and Non-Runners (Controls)

Group	$30,000+	$25-30,000	$20-25,000	$15-20,000	< $15,000
			Income Bracket		
University	-	-	-	-	10
70+	5	-	-	-	1
40-69	7	4	1	-	3
20-39	14	3	-	-	1
Controls	1	-	2	2	13
Total Runners	26	7	2	2	13
Total Controls	1	-	2	2	13

Table 5. Educational Achievement Levels

Group	High School	Bachelor Degree	Masters	Ph.D./M.D.	Other Professional Qualifications
			Education Level Limit		
University	4	3	1	-	2
70+	1	3	-	2	-
40-69	2	4	3	3	4
20-39	3	6	3	3	3
Controls	8	6	3	1	-
Total Runners	10	16	7	8	7
Total Controls	8	6	3	1	-

Table 6. External Achievement Motivation: Parental Encouragement

Group	Parental Encouragement	
	Yes	No
University	9	1
70+	4	2
40-69	13	2
20-39	16	2
Controls	17	1
Total Runners	42	7
Total Controls	17	1

Table 7. Number of Years Engaged in Serious Running

Group	Mean Number of Years	N
University	6.3	10
70+	13.1	6
40-69	15.2	15
20-39	11.3	18
Grand Mean ($\bar{X}$)	11.4	49

APPENDIX 2(b)

PONDURAL INDEX - MEANS, STANDARD DEVIATIONS AND STANDARD ERROR

Group		5 Groups	4 Groups	3 Groups
1	$\overline{X}$	13.08	12.927	13.02
	SD	0.6107	0.2857	0.3959
	SE	0.215	0.031	0.077
2	$\overline{X}$	12.84	13.079	12.78
	SD	0.409	0.445	0.532
	SE	0.18	0.107	0.125
3	$\overline{X}$	13.087	12.78	12.918
	SD	0.3928	0.532	0.3946
	SE	0.039	0.125	0.118
4	$\overline{X}$	12.716	12.918	
	SD	0.45	0.3946	
	SE	0.11	0.118	
5	$\overline{X}$	12.918		
	SD	0.3946		
	SE	0.118		

Group 1 Univ.
Group 2 70+ mpw
Group 3 40-69 mpw
Group 4 20-39 mpw
Group 5 Control

Group 1 70+ mpw
Group 2 40-69 mpw
Group 3 20-39 mpw
Group 4 Control

Group 1 40+ mpw
Group 2 20-39 mpw
Group 3 Control

APPENDIX 3

TABULATION OF RESPONSES FROM RUNNING COMMITMENT QUESTIONNAIRE

Tables entered as frequency tables by question order:

#1. Mileage Categories

Mileage Categories Miles per Week	Club Runners Frequency	University Runners	Total
20-39	18	3	21
40-69	15	2	17
70+	6	4	10

#2. Number of Runs per Week

Frequency (Runs per Week)

Group	5	6	7	8	9	10	11	12	13	14	Mean
University	-	2	5	-	-	1	-	-	-	2	8.5
70+	-	-	2	-	1	-	2	1	-	2	8.5
40-69	2	7	3	1	-	1	-	-	1	-	6.9
20-39	14	3	1	-	-	-	-	-	-	-	5.0
Total	16	12	11	1	1	2	2	1	1	2	

#3. Frequency of Competition

F/Response

Group	Never	Occasionally	Frequently	Every Opportunity
University			8	2
70+			6	
40-69	1	10	3	1
20-39		11	7	
Total	1	21	24	3

#4. Period of Day When Running

Group	Early Morning	Between Breakfast/ Lunch	Between Lunch/ Supper	After Supper	More Than Once
University	6		9	1	5
70+	3	2	4		2
40-69	3	7	9		3
20-39	1	8	8	2	1
Total	13	17	30	3	11

#5. Frequency of Missing Meals and Running

Group	Miss Breakfast	Miss Lunch	Miss Supper	Never Miss Meal	Occasionally Miss Meal	Frequently Miss Meal
University	-	-	1	3	5	1
70+	2	2	1	2	1	2
40-69		4	-	1	10	-
20-39	2	7	1	2	11	1
Total	4	13	2	8	27	4

#6. Running and Lateness

Group	Never	Occasionally	Often
University	6	4	
70+	6		
40-69	6	7	2
20-39	11	6	1
Total	29	17	3

#7. Number of Meals Eaten per Day

Group	One	Two	Three	More than Three
	Meals per Day			
University		5	5	-
70+		3	3	-
40-69	2	3	10	-
20-39	1	6	11	-
Total	3	17	29	0

#8. Running and Domestic Responsibility

Group	Never	Occasionally	Often
	Neglect Domestic Responsibility		
University	1	9	0
70+	0	4	2
40-69	5	9	1
20-39	4	13	1
Total	10	35	4

#9. Significance of Running in Life

Group	Hobby	Enjoyment	Fundamental Important Aspect of Daily Life	Losing Weight	Gain Fitness
	Degree of Significance				
University	-	2	5	1	2
70+	-	-	5	-	-
40-69	1	5	4	-	5
20-39	1	2	9	1	5
Total	2	7	23	2	12

#10. Response to Doctor's Order to Cease Running

	Response Category		
Group	Obey	Rest 1 Week	Ignore
University	2	7	1
70+	-	3	2
40-69	2	12	1
20-39	3	13	2
Total	7	35	6

#11. Emotions Related to Omission of Daily Run

	Emotional Response			
Group	Indifferent	Mildly Regretful	Anxious	Extremely Anxious
University	2	6	1	1
70+	-	1	4	-
40-69	1	7	7	1
20-39	1	12	5	-
Total	4	26	17	1

#12. Need to Run Following Ingestion of Food

	Need to Run Following Meal		
Group	Always	Sometimes	Never
University	-	2	8
70+	-	1	4
40-69	-	2	13
20-39	-	5	13
Total	-	10	38

#13. Immediate Reaction to Knowledge of Need to Cease Running

Major Response Category	Frequency (All Groups)
Ignore	8
Find Alternate Form of Exercise	20
Disbelief	4
Intense Depression and Feeling of Loss	14
Loss of Training and Fitness	1
Increased Weight and Need to Diet	2
Total	49

APPENDIX 4

BODY IMAGE PERCEPTION 5 GROUP ANALYSIS - MEANS, STANDARD DEVIATIONS AND STANDARD ERROR

		Shoulder		Waist		Hip		Mean		Height	
		1	2	1	2	1	2	1	2	1	2
Group 1	$\bar{X}$	88.80*	89.6	109.1	121.75	105.6	110.6	101.15	107.41	74.25	77.54
	SD	14.07	10.45	19.23	21.37	25.40	22.80	15.72	15.50	13.95	19.63
	SE	4.45	3.48	6.08	7.12	8.04	7.62	4.07	5.17	4.41	6.54
Group 2	$\bar{X}$	118.9*	111.7	123.3	121.1	118.2	132.6	120.15	121.8	78.20	80.88
	SD	31.60	26.81	17.19	23.90	19.0	40.81	20.71	27.58	11.29	7.53
	SE	12.89	10.94	7.02	9.75	7.75	16.6	8.45	11.26	4.60	3.07
Group 3	$\bar{X}$	104.20	99.23	136.96	131.50	125.70	120.75	123.90	117.60	74.74	74.40
	SD	20.56	12.28	39.34	38.90	28.13	24.20	27.40	22.90	15.10	14.40
	SE	5.30	3.28	10.15	10.39	7.26	6.46	7.07	6.13	3.89	3.85
Group 4	$\bar{X}$	99.2	97.9	111.24	112.5	114.8	118.5	108.5	109.6	72.9	71.2
	SD	20.25	4.77	21.69	22.8	19.76	21.82	17.82	19.14	8.82	10.80
	SE	19.55	4.88	5.11	5.70	4.66	5.45	4.20	4.18	2.20	2.7
Group 5	$\bar{X}$	95.60	92.60	117.02	105.40	117.93	111.14	110.84	102.99	74.00	73.38
	SD	14.70	14.00	29.11	22.27	26.80	20.69	19.48	16.23	11.80	10.75
	SE	3.48	3.75	6.87	5.95	6.33	5.52	4.59	4.33	2.70	2.87
Total	$\bar{X}$	99.66	97.10	119.31	117.69	117.00	117.52	112.50	110.84	74.34	74.40
	SD	20.36	17.05	29.13	28.17	24.68	24.69	21.56	20.28	12.02	13.02
	SE	2.48	2.22	3.55	3.66	3.01	3.21	2.63	2.64	1.49	1.69

* Denotes significant difference: Group 1 vs. 2, $p < 0.1$

APPENDIX 4

BODY IMAGE PERCEPTION 4 GROUP ANALYSIS - MEANS, STANDARD DEVIATIONS AND STANDARD ERROR

		Shoulder		Waist		Hip		Mean		Height	
		1	2	1	2	1	2	1	2	1	2
Group 1	$\bar{X}$	105.5	99.84	122.86	122.23	133.11	122.05	113.83	114.70	80.48	81.83
	SD	29.46	25.44	16.21	23.32	22.13	38.96	19.29	25.78	11.53	7.13
	SE	9.31	8.04	5.12	7.37	6.99	12.32	6.10	8.15	3.64	2.55
Group 2	$\bar{X}$	102.02	99.05	129.73	130.5	122.13	119.1	119.40	116.56	74.25	76.46
	SD	21.09	11.50	39.53	36.56	28.53	23.04	27.75	21.10	14.16	16.50
	SE	4.97	2.87	9.31	9.14	6.72	9.76	6.54	5.40	3.34	4.12
Group 3	$\bar{X}$	97.99	97.29	110.64	113.59	113.70	118.5	107.50	109.80	71.50	69.53
	SD	19.35	18.38	21.05	22.82	20.98	20.21	17.83	18.10	9.83	12.28
	SE	4.22	4.21	4.59	5.23	4.58	4.63	3.89	4.15	2.25	2.81
Group 4	$\bar{X}$	95.60	92.58	117.02	105.38	117.94	111.14	110.84	102.90	74.01	73.38
	SD	14.77	14.04	29.17	22.27	26.87	20.69	19.50	16.23	11.80	10.75
	SE	3.48	3.75	6.87	5.95	6.34	5.53	4.60	4.33	2.78	2.87
Total	$\bar{X}$	99.56	97.1	119.31	117.69	117.02	117.52	112.54	110.85	74.34	74.4
	SD	20.36	17.05	29.13	28.17	24.68	24.69	21.6	20.28	12.02	13.02
	SE	2.48	2.22	3.55	3.68	3.01	3.21	2.63	2.64	1.49	1.69

APPENDIX 4

BODY IMAGE PERCEPTION 3 GROUP ANALYSIS - MEANS, STANDARD DEVIATIONS AND STANDARD ERROR

		Shoulder		Waist		Hip		Mean		Height	
		1	2	1	2	1	2	1	2	1	2
Group 1	$\bar{X}$	103.27	99.36	127.28	127.32*	118.91	120.21	117.4	115.85	76.47	78.52
	SD	23.92	17.68	32.90	31.85	26.36	29.45	24.82	22.81	13.40	13.73
	SE	4.52	3.46	6.21	6.24	4.98	5.77	4.69	4.47	2.53	2.69
Group 2	$\bar{X}$	97.99	97.30	110.65	113.59	113.70	118.52	107.51	109.80	71.50	69.53
	SD	19.35	18.40	21.04	22.82	20.98	20.21	17.83	18.10	9.83	12.28
	SE	4.22	4.21	4.59	5.23	4.58	4.63	3.89	4.15	2.25	2.87
Group 3	$\bar{X}$	95.60	92.60	117.02	105.38*	117.93	111.14	110.84	102.9	74.01	73.38
	SD	14.77	14.04	29.17	22.27	26.87	20.69	19.5	16.23	11.80	10.75
	SE	3.48	3.75	6.87	5.15	6.33	5.52	4.59	4.33	2.75	2.87
Total	$\bar{X}$	99.56	97.1	119.31	117.7	117.02	117.52	112.54	110.84	74.34	74.40
	SD	20.36	17.05	29.13	28.17	24.68	24.69	21.56	20.28	12.02	13.02
	SE	2.48	2.22	3.55	3.67	3.01	3.21	2.63	2.64	1.49	1.69

* Denotes significant difference: Group 1 vs. 3, $p < 0.05$

APPENDIX 5

EATING ATTITUDE TEST 5, 4, AND 3 GROUP ANALYSIS - MEAN, STANDARD DEVIATIONS AND STANDARD ERROR

Group		5 Group Analysis	4 Group Analysis	3 Group Analysis
	$\overline{X}$	10.10	11.30	9.60
1	SD	5.34	5.10	4.50
	SE	1.69	1.61	0.84
	$\overline{X}$	9.83	8.67	10.43
2	SD	3.12	3.96	5.98
	SE	1.27	0.93	1.30
	$\overline{X}$	8.73	10.43	7.38
3	SD	4.31	5.98	5.10
	SE	1.14	1.30	1.20
	$\overline{X}$	10.94	7.38	
4	SD	6.23	5.10	
	SE	1.46	1.20	
	$\overline{X}$	7.38		
5	SD	5.10		
	SE	1.20		
	$\overline{X}$	9.27	9.27	9.27
Total	SD	5.21	5.21	5.21
	SE	0.63	0.63	0.63

Analysis of variance revealed no differences among groups at any level of significance.

APPENDIX 6

PERSONALITY MEASURES OF 5 GROUP ANALYSIS - MEANS, STANDARD DEVIATIONS AND STANDARD ERRORS

Group		Anx	BDI	Complex	Conform	Ener Level	Indiv	Inter Per	Organ	Respons	Risk	Self	Social Ad	Social Fr	Toler	Value	Infreq
	$\bar{X}$	50.1	48.5	44.8	52.8	49.5	44.5	43.60	48.3	43.50**	51.10	46.4	52.2	49.1	46.10	52.7	51.10
1	SD	12.5	12.82	13.4	10.14	13.4	18.12	6.86	8.43	10.12	10.90	12.08	12.83	10.72	12.87	9.93	11.32
	SE	3.97	4.05	4.24	3.20	4.23	5.73	2.17	2.67	3.20	3.45	3.82	4.05	3.39	4.07	3.14	3.58
	$\bar{X}$	39.7	47.0	47.0	46.17	57.0	49.83	40.87	58.83	58.3**	40.67	56.83	40.0	43.3	45.33	55.5	57.3
2	SD	4.41	12.5	7.51	5.5	4.86	13.54	11.34	7.02	5.88	8.38	9.92	9.79	7.03	10.23	6.62	12.04
	SE	1.8	5.10	3.06	2.24	1.98	5.52	4.63	2.87	2.40	3.42	4.05	4.0	2.87	4.17	2.70	4.91
	$\bar{X}$	49.46	49.4	43.0	48.7	50.26	46.87	48.9	51.80	56.86**	44.20	51.80	45.87	46.6	49.33	54.6	54.2
3	SD	11.09	8.47	12.0	9.1	8.87	12.66	8.87	8.98	7.63	10.41	12.19	11.78	6.31	10.03	10.12	5.75
	SE	2.86	2.18	3.09	2.34	2.29	3.26	2.29	2.32	1.97	2.68	3.14	3.04	1.62	2.59	2.61	1.48
	$\bar{X}$	47.11	47.77	43.0	49.4	55.17*	48.11	48.77	52.33	56.5**	44.27	48.2	44.0	45.4	48.77	56.24	49.0
4	SD	11.18	9.25	9.81	10.7	8.08	10.67	7.5	7.98	8.19	9.56	12.36	9.87	9.18	8.0	11.51	8.61
	SE	2.63	2.18	2.31	2.52	1.90	2.51	1.77	1.88	1.93	2.25	2.91	2.32	2.16	1.88	2.71	2.03
	$\bar{X}$	50.83	45.94	44.03	51.2	45.72*	45.0	47.3	46.83	52.3	44.67	48.2	46.4	49.5	43.78	52.2	47.0
5	SD	11.98	8.17	9.72	8.56	9.68	12.58	12.53	12.03	8.02	9.12	10.83	11.50	9.9	7.98	9.5	4.36
	SE	2.82	1.92	2.29	2.01	2.28	2.96	2.95	2.83	1.89	2.15	2.55	2.70	2.33	1.88	2.23	1.02
	$\bar{X}$	48.41	47.68	43.91	49.55	50.85	46.38	46.9	50.71	53.70	45.6	49.52	45.92	47.2	46.85	54.24	50.68
Total	SD	11.31	9.90	10.48	9.26	9.94	12.87	9.74	9.81	9.24	9.85	11.70	11.33	8.86	9.49	9.94	8.41
	SE	1.38	1.16	1.28	1.13	1.22	1.57	1.19	1.19	1.13	1.20	1.42	1.38	1.08	1.15	1.21	1.02

* Denotes pairs significant difference at 0.01

** Denotes pairs significant difference at 0.05

APPENDIX 6

PERSONALITY MEASURES OF 4 GROUP ANALYSIS - MEANS, STANDARD DEVIATIONS AND STANDARD ERRORS

Group		Anx	BDI	Complex	Conform	Ener Level	Indiv	Inter Per	Organ	Respons	Risk	Self	Social Ad	Social Fr	Toler	Value	Infreq
	$\bar{X}$	42.50*	46.80	46.30	47.50	54.80	51.40	41.90	53.90	51.60	45.90	54.50	45.60	47.10	44.20	54.60	56.4**
1	SD	10.3	13.38	7.93	8.42	9.25	10.74	9.53	9.23	13.36	11.62	9.47	13.46	12.04	14.20	7.44	11.01
	SE	3.26	4.23	2.50	2.66	2.92	3.39	3.01	2.91	4.22	3.67	2.99	4.26	3.80	4.49	2.35	3.48
	$\bar{X}$	49.3	49.5	42.83	48.83	51.89	43.72	47.4	52.4	54.22	45.61	51.11	46.55	46.4	49.11	53.72	53.0
2	SD	10.67	8.07	12.70	8.30	9.04	14.48	9.12	8.32	9.35	10.98	12.01	11.86	5.83	9.36	10.08	6.17
	SE	2.51	1.90	2.99	1.96	2.13	3.63	2.15	1.96	2.20	3.58	2.83	2.79	1.37	2.20	2.37	1.45
	$\bar{X}$	46.38	47.04	43.57	51.0	52.47	47.8	48.47	51.04	55.42	46.23	46.90	45.14	46.0	48.80	56.23	49.14
3	SD	11.5	9.93	10.63	11.12	10.33	12.16	7.26	8.70	8.10	9.22	12.86	10.45	8.66	7.56	1.39	9.73
	SE	2.50	2.16	2.32	2.42	2.25	2.65	1.58	1.90	1.76	2.01	2.80	2.28	1.88	1.64	2.48	2.12
	$\bar{X}$	50.83	45.9	44.05	51.2	45.72	45.0	47.3	46.83	52.3	44.66	48.2	46.38	49.5	43.77	52.2	47.0**
4	SD	11.98	8.17	9.72	8.56	9.68	12.58	12.53	12.03	8.02	9.12	10.8	11.49	9.9	7.98	9.50	4.38
	SE	2.82	1.92	2.29	2.01	2.28	2.96	2.95	2.83	1.89	2.15	2.55	2.70	2.33	1.88	2.23	1.02
	$\bar{X}$	48.41	47.7	43.91	49.95	50.85	46.38	46.91	50.71	53.70	45.59	49.52	45.92	47.22	46.85	54.23	50.68
Total	SD	11.31	9.50	10.48	9.26	9.99	12.87	9.74	9.81	9.24	9.85	11.7	11.33	8.86	9.49	9.94	8.41
	SE	1.38	1.16	1.28	1.13	1.22	1.57	1.19	1.19	1.13	1.20	1.42	1.38	1.082	1.15	1.21	1.02

* Denotes significant difference, $p < 0.1$

** Denotes significant difference, $p < 0.05$

APPENDIX 6

PERSONALITY MEASURES OF 3 GROUP ANALYSIS - MEANS, STANDARD DEVIATIONS AND STANDARD ERRORS

Group		Anx	BDI	Complex	Conform	Ener Level	Indiv	Inter Per	Organ	Respons	Risk	Self	Social Ad	Social Fr	Toler	Value	Infreq
	$\bar{X}$	46.09	46.29	44.07	48.35	52.42*	46.46	45.46	52.96	53.28	45.71	52.32	46.21	46.67	47.35	54.03	54.21**
1	SD	10.87	10.04	11.20	8.21	9.05	13.92	9.49	8.51	10.78	11.00	11.11	12.22	8.35	11.32	9.09	8.19
	SE	2.05	1.88	2.11	1.55	1.71	2.63	1.79	1.60	2.03	2.07	2.10	2.30	1.57	2.14	1.71	1.54
	$\bar{X}$	48.38	47.04	43.57	51.0	52.47	47.47	48.47	51.04	55.42	46.23	46.90	45.14	46.0	48.80	56.2	49.14*
2	SD	11.5	9.93	10.63	11.12	10.33	12.16	7.25	8.70	8.10	9.22	12.86	10.46	8.66	7.56	11.4	9.73
	SE	2.50	2.16	2.32	2.42	2.25	2.65	1.58	1.90	1.76	2.01	2.80	2.28	1.88	1.64	2.48	2.12
	$\bar{X}$	50.83	46.94	44.05	51.2	46.72*	45.0	47.3	46.83	52.33	44.67	48.22	46.38	49.5	43.77	52.2	47.0**
3	SD	11.98	8.17	9.72	8.94	9.68	12.58	12.53	12.03	8.02	9.12	10.83	11.49	9.90	7.98	9.50	4.36
	SE	2.82	1.92	2.29	2.01	2.28	2.96	2.95	2.83	1.89	2.15	2.55	2.70	2.33	1.88	2.23	1.03
	$\bar{X}$	48.41	47.68	43.91	49.95	50.85	46.4	46.91	50.71	53.70	45.59	49.52	45.92	47.22	46.85	54.23	50.7
Total	SD	11.31	9.50	10.48	9.26	9.99	12.87	9.74	9.81	9.24	9.85	11.70	11.22	8.86	9.49	9.94	8.41
	SE	1.38	1.1616	1.28	1.13	1.22	1.57	1.19	1.19	1.13	1.20	1.42	1.38	1.08	1.15	1.21	1.02

* Denotes significant difference, $\alpha = 0.1$

** Denotes significant difference, $\alpha = 0.05$

APPENDIX 7(a)

HORMONE LEVELS 5 GROUP ANALYSIS - MEANS, STANDARD DEVIATIONS AND STANDARD ERROR

		LH	FSH	T	Non SHBG Bound T	Free T	SHBG	TSH	Cortisol	T_4	T_3U	FT_4I	Prolactin
Group 1	$\bar{X}$	8.331	5.95	6.51	283.5	10.31	4.83*	4.9170	12.17	106.08	44.39	94.41	5.94
	SD	3.90	3.61	1.71	117.76	4.21	0.59	0.9612	7.36	35.85	6.47	18.64	2.03
	SE	1.23	1.44	0.54	37.23	1.33	0.18	0.3040	2.33	11.33	2.04	5.89	0.64
Group 2	$\bar{X}$	10.5	8.82	6.42	216.3	7.91	5.76	5.3083	13.99	95.88	41.45	83.35	6.86
	SD	5.08	7.86	1.4469	66.74	2.42	0.83	1.02	5.87	21.27	2.34	14.93	2.90
	SE	2.07	3.20	0.5907	27.24	0.99	0.34	0.42	2.39	8.68	0.95	6.09	3.30
Group 3	$\bar{X}$	9.0	6.20	7.24	216.16**	9.36	5.96*	4.7533	12.07	193.91	40.81	94.31	5.75
	SD	5.14	4.71	2.33	94.97	4.53	0.97	0.9522	4.64	331.88	4.33	18.07	2.54
	SE	1.32	1.21	0.60	24.52	1.17	0.25	0.245	1.19	85.69	1.11	4.66	0.66
Group 4	$\bar{X}$	7.33	10.57	7.12	267.928	9.77	5.64	6.05	15.02	106.39	40.99	91.95	6.25
	SD	2.917	10.47	2.18	131.44	4.76	0.85	2.42	6.29	23.12	3.80	18.15	2.44
	SE	0.7797	2.80	0.58	35.14	1.27	0.23	0.67	1.68	6.18	1.01	4.85	0.65
Group 5	$\bar{X}$	10.10	5.03	8.25	387.31**	15.15	5.11	4.79	16.56	114.68	39.69	95.20	8.08
	SD	4.17	3.10	2.78	214.07	9.65	0.72	0.67	8.01	26.79	4.06	17.17	1.97
	SE	1.04	0.77	0.69	53.51	2.41	0.1811	0.18	2.0	6.69	1.01	4.29	0.52
Total	$\bar{X}$	8.94	7.11	7.28	284.0	10.99	5.46	5.14	14.13	129.00	41.21	92.94	8.83
	SD	4.25	6.56	2.3	155.8	6.54	0.89	1.43	6.64	166.28	4.55	17.43	3.45
	SE	0.84	0.8412	0.29	19.94	0.83	0.11	0.18	0.85	21.29	0.58	2.23	0.44

** Denotes significant difference, $p < 0.05$

* Denotes significant difference, $p < 0.1$

APPENDIX 7(a)

HORMONE LEVELS 4 GROUP ANALYSIS - MEANS, STANDARD DEVIATIONS AND STANDARD ERROR

		LH	FSH	T	Non SHBG Bound T	Free T	SHBG	TSH	Cortisol	T_4	T_{3u}	FT_4I	Prolactin
Group 1	$\bar{X}$	10.56	8.45	6.61	258.0	9.39	5.27	5.08	14.81	98.59	42.30	87.49	6.81
	SD	4.18	6.27	1.26	97.16	3.46	0.911	0.895	7.40	20.40	3.53	13.23	2.55
	SE	1.32	1.98	0.4	30.72	1.09	0.28	0.28	2.34	6.33	1.11	4.18	2.02
Group 2	$\bar{X}$	9.11	6.2	7.34	240.02**	9.97	5.78	4.73	11.87	111.19	41.46	94.93	5.70
	SD	4.77	4.45	2.14	104.52	4.42	1.04	0.87	4.38	29.40	4.77	19.95	2.45
	SE	1.12	1.05	0.50	24.03	1.04	0.24	0.20	1.03	6.93	1.12	4.70	0.57
Group 3	$\bar{X}$	6.71	9.26	6.69	248.62	9.08**	5.56	5.93	13.83	105.65	41.72	91.91	6.10
	SD	3.02	9.91	2.27	128.62	4.66	0.79	2.26	6.48	26.12	5.26	17.65	2.35
	SE	0.73	2.40	0.55	31.19	1.13	0.19	0.56	1.57	6.33	1.27	4.28	0.57
Group 4	$\bar{X}$	10.10	5.03	8.25	387.31**	15.15**	5.11	4.79	16.55	114.68	39.69	95.20	8.008
	SD	4.17	3.10	2.78	214.07	9.65	0.72	0.67	8.01	26.79	4.06	17.17	1.97
	SE	1.04	0.77	0.69	53.51	2.41	0.18	0.81	2.00	6.69	1.01	4.29	0.52
Total	$\bar{X}$	8.94	7.11	7.28	284.00	15.15	5.46	5.14	14.13	108.49	41.21	92.94	6.83
	SD	4.25	6.56	2.30	155.80	9.65	0.89	1.43	6.64	26.41	4.55	17.43	3.45
	SE	0.54	0.84	0.29	19.94	2.41	0.11	0.18	0.85	3.38	0.58	2.23	0.44

** Denotes significant difference, $p < 0.05$

APPENDIX 7(a)

HORMONE LEVELS 3 GROUP ANALYSIS - MEANS, STANDARD DEVIATIONS AND STANDARD ERROR

		LH	FSH	T	Non SHBG Bound T	Free T	SHBG	TSH	Cortisol	T_4	T_{3U}	FT_4I	Prolactin
Group 1	$\bar{X}$	9.63*	7.0	7.08	246.44**	9.76**	5.60	4.85	12.92	106.69	41.76	92.27	6.07**
	SD	4.54	5.18	1.88	100.51	4.04	1.01	0.88	5.69	26.75	4.32	17.95	2.50
	SE	0.85	0.97	0.35	18.99	0.76	0.19	0.16	1.07	5.05	0.81	3.39	0.83
Group 2	$\bar{X}$	6.71*	9.26	6.69	248.62**	9.08**	5.56	5.93	13.83	105.65	41.72	91.91	5.70**
	SD	3.02	9.91	2.27	128.62	4.66	0.79	2.26	6.48	26.12	5.26	17.65	2.45
	SE	0.73	2.40	0.55	31.19	1.13	0.19	0.56	1.57	6.33	1.27	4.28	0.57
Group 3	$\bar{X}$	10.10*	5.03	8.25	387.31**	15.15**	5.11	4.79	16.55	114.68	39.69	95.20	8.008**
	SD	4.17	3.10	2.78	214.07	9.65	0.72	0.67	8.01	26.79	4.06	17.17	1.97
	SE	1.04	0.77	0.69	53.5	2.41	0.18	0.18	2.00	6.69	1.01	4.29	0.52
Total	$\bar{X}$	8.94	7.11	7.28	284.00	10.99	5.46	5.14	14.13	108.5	41.21	92.94	6.83
	SD	4.25	6.56	2.30	155.8	6.54	0.89	1.43	6.64	26.41	4.55	17.43	3.45
	SE	0.54	0.84	0.29	19.94	0.83	0.11	0.18	0.85	3.38	0.58	2.23	0.44

** Denotes significant difference, $p < 0.05$

* Denotes significant difference, $p < 0.1$

APPENDIX 7(b)

SUMMARY TABLE OF SIGNIFICANT DIFFERENCES ($p < 0.05$) AMONG GROUPS: HORMONE LEVELS

Hormone	5 Group Analysis		4 Group Analysis		3 Group Analysis	
	Group	Significance Level	Group	Significance Level	Group	Significance Level
Non SHBG Bound T	3 vs 5	$p < 0.05$	2 vs 4	$p < 0.05$	1 vs 3	$p < 0.05$
					2 vs 3	$p < 0.05$
Free T					1 vs 3	$p < 0.05$
					2 vs 3	$p < 0.05$
SHBG	1 vs 3	$p < 0.05$				
Prolactin					1 vs 3	$p < 0.05$
					2 vs 3	$p < 0.05$

APPENDIX 8(a)

SPEARMAN CORRELATION MATRIX

40 MILES/WEEK RUNNERS (N=28)

	Waist	Mean	EAT	Anx	Self	Infreq	Total T	Free T	Non SHBG T	Prol	LH	FSH	Cort
Waist	1.00	+0.937	+0.156	+0.410	-0.338	+0.414	+0.192	-0.0673	-0.274	+0.844	-0.077	-0.138	-0.051
Mean	+0.937	1.00	+0.055	+0.050	-0.172	+0.371	+0.286	-0.009	-0.076	+0.153	-0.034	-0.284	+0.075
EAT	+0.156	+0.055	1.00	+0.072	-0.031	+0.103	+0.044	+0.19	+0,194	-0.331	-0.427	-0.035	-0.635
Anx	+0.419	+0.05	+0.072	1.00	-0.396	-0.016	+0.047	-0.043	-0.383	-0.2711	-0.213	-0.131	-0.139
Self	-0.338	-0.172	-0.031	-0.396	1.00	-0.018	+0.016	-0.074	+0.066	+0.198	-0.186	+0.217	+0.06
Infreq	+0.419	+0.371	+0.1303	-0.016	-0.018	1.00	+0.176	-0.872	-0.247	-0.259	+0.073	+0.046	-0.078
Total T	+0.192	+0.286	+0.044	+0.097	+0.016	+0.176	1.00	+0.684	+0.601	+0.199	+0.204	+0.229	+0.334
Free T	-0.067	-0.009	+0.19	-0.043	-0.074	-0.872	+0.684	1.00	+0.987	+0.044	+0.020	-0.092	+0.195
Non SHBG T	-0.274	-0.076	+0.194	-0.383	+0.066	-0.247	+0.601	+0.987	1.00	-0.026	+0.830	-0.016	+0.157
Prol	+0.844	+0.153	-0.331	-0.2711	+0.198	-0.259	+0.199	+0.044	-0.026	1.00	+0.511	+0.517	+0.365
LH	-0.077	-0.034	-0.427	-0.213	-0.186	+0.073	+0.204	+0.020	+0.830	+0.511	1.00	+0.376	+0.228
FSH	-0.138	-0.284	-0.035	-0.131	+0.217	+0.046	+0.229	-0.092	-0.016	+0.517	+0.376	1.00	+0.278
Cort	-0.051	+0.075	-0.635	-0.139	+0.06	-0.078	+0.334	+0.195	+0.157	+0.365	+0.228	+0.278	1.00

APPENDIX B(a)

SPEARMAN CORRELATION MATRIX
20-40 MILES/WEEK RUNNERS (N = 21)

	Waist	Mean	EAT	Anx	Self	Infreq	Total T	Free T	Non SHBG T	Prol	LH	FSH	Cort
Waist	1.00	+0.747	-0.230	-0.017	-0.046	+0.261	-0.910	-0.778	-0.853	+0.686	+0.159	-0.613	-0.839
Mean	+0.747	1.00	-0.084	-0.154	-0.143	+0.039	-0.796	-0.662	-0.565	-0.491	+0.08	-0.650	-0.608
EAT	-0.230	-0.084	1.00	+0.292	-0.057	-0.216	-0.222	-0.346	-0.530	-0.05	-0.033	+0.101	-0.075
Anx	-0.017	-0.154	+0.292	1.00	-0.270	+0.204	+0.116	+0.092	+0.016	+0.205	-0.003	-0.011	-0.254
Self	-0.046	-0.143	-0.057	-0.270	1.00	-0.050	-0.048	-0.503	-0.204	+0.224	-0.658	-0.537	+0.212
Infreq	+0.261	+0.039	-0.216	+0.204	-0.050	1.00	-0.294	+0.08	-0.120	-0.186	+0.209	-0.343	-0.248
Total T	-0.910	-0.796	-0.222	+0.116	-0.048	-0.294	1.00	+0.8206	+0.968	+0.262	+0.184	+0.211	+0.431
Free T	-0.778	-0.662	-0.346	+0.092	-0.503	+0.08	+0.863	1.00	+0.972	+0.1103	+0.117	+0.009	+0.228
Non SHBG T	-0.853	-0.566	-0.53	+0.016	-0.204	-0.120	+0.968	+0.972	1.00	+0.211	+0.123	+0.266	+0.344
Prol	+0.686	-0.491	-0.05	+0.205	+0.224	-0.186	+0.262	+0.110	+0.211	1.00	-0.107	+0.293	+0.444
LH	+0.159	+0.08	-0.033	-0.003	-0.658	+0.209	+0.184	+0.117	+0.123	-0.107	1.00	+0.276	-0.074
FSH	-0.613	-0.650	+0.101	-0.011	-0.537	-0.343	+0.211	+0.009	+0.266	+0.293	+0.276	1.00	+0.206
Cort	-0.839	-0.608	-0.075	-0.254	+0.212	-0.248	+0.431	+0.228	+0.344	+0.444	-0.074	+0.206	1.00

APPENDIX B(a)

SPEARMAN CORRELATIONS MATRIX
CONTROLS (N = 18)

	Waist	Mean	EAT	Anx	Self	Infreq	Total T	Free T	Non SHBG T	Prol	LH	FSH	Cort
Waist	1.00	+0.902	+0.1367	+0.052	-0.295	+0.462	-0.077	+0.304	+0.251	+0.796	+0.227	+0.004	+0.03
Mean	+0.902	1.00	+0.231	+0.120	-0.106	+0.327	+0.178	+0.173	+0.078	+0.133	+0.600	+0.075	+0.051
EAT	+0.1367	+0.231	1.00	+0.013	+0.04	+0.059	+0.15	-0.084	-0.13	-0.194	+0.031	+0.031	-0.086
Anx	+0.052	+0.120	+0.013	1.00	-0.112	+0.1414	-0.214	-0.0393	+0.173	-0.281	-0.219	-0.059	+0.118
Self	-0.295	-0.106	+0.04	-0.112	1.00	-0.025	+0.187	-0.172	-0.08	-0.755	-0.167	+0.168	+0.424
Infreq	+0.462	+0.327	+0.059	+0.1414	-0.025	1.00	-0.30	-0.288	-0.230	-0.367	+0.429	+0.184	+0.036
Total T	-0.077	+0.178	+0.15	-0.214	+0.187	-0.30	1.00	+0.9206	+0.9089	+0.095	-0.480	+0.088	-0.144
Free T	+0.304	+0.173	-0.084	-0.0393	-0.172	-0.288	+0.9206	1.00	+0.971	+0.001	-0.517	-0.023	-0.04
Non SHBG T	+0.251	+0.078	-0.13	+0.173	-0.08	-0.230	+0.9084	+0.971	1.00	+0.192	-0.468	-0.266	+0.113
Prol	+0.796	+0.133	-0.194	-0.281	-0.755	-0.367	+0.095	+0.001	+0.192	1.00	-0.175	+0.094	-0.396
LH	+0.227	+0.060	-0.470	-0.219	-0.167	+0.429	-0.480	-0.517	-0.468	-0.175	1.00	+0.289	-0.136
FSH	+0.004	+0.075	+0.031	-0.054	+0.168	+0.184	+0.088	-0.023	-0.266	+0.094	+0.289	1.00	-0.119
Cort	+0.03	+0.051	-0.086	+0.118	+0.424	+0.036	-0.144	-0.04	+0.113	-0.396	-0.136	-0.119	1.00

APPENDIX 8(b)

SIGNIFICANT SPEARMAN RANK ORDER CORRELATIONS
20-40 MILES PER WEEK RUNNERS N = 21

	Waist	Mean	EAT	Anx	Self	Infreq	Total T	Free T	Non-SHBG T	Prol	LH	FSH	Cort
Waist	1.00	+0.747 **					-0.910 **	-0.778 **	-0.853 **	+0.686 **		-0.613 **	-0.839 **
Mean		1.00					-0.796 **	-0.662 **	-0.566 *	-0.491 *		-0.650 **	-0.608 **
EAT			1.00						-0.530 **				
Anx				1.00									
Self					1.00			-0.503 *			-0.650 **	-0.537 *	
Infreq						1.00							
Total T							1.00	+0.920 **	+0.968 **				+0.431 *
Free T								1.00	+0.972 **				
Non SHBG T									1.00				
Prol										1.00			
LH											1.00		
FSH												1.00	
Cort													1.00

* Denotes significance, $p < 0.05$

** Denotes significance, $p < 0.01$

APPENDIX 8(b)

SIGNIFICANT SPEARMAN RANK ORDER CORRELATIONS
40+ MILES PER WEEK RUNNERS N = 28

	Waist	Mean	EAT	Anx	Self	Infreq	Total T	Free T	Non-SHBG T	Prol	LH	FSH	Cort
Waist	1.00	+0.937 **		+0.410 *	-0.338 *	+0.419 *				+0.844 **			
Mean		1.00				+0.371 *							
EAT			1.00							-0.331 *	-0.427 *		-0.635 **
Anx				1.00	-0.396 *				-0.383 *				
Self					1.00								
Infreq						1.00		-0.872 **					
Total T							1.00	+0.684 **	+0.601 **				
Free T								1.00	+0.987 **				
Non-SHBG T									1.00		+0.83 **		
Prol										1.00	+0.511 **	+0.517 **	+0.365 *
LH											1.00	+0.376 *	
FSH												1.00	
Cort													1:00

* Denotes significance, $p < 0.05$

** Denotes significance, $p < 0.01$

APPENDIX B(b)

SIGNIFICANT SPEARMAN RANK ORDER CORRELATIONS
CONTROL GROUP N = 18

	Waist	Mean	EAT	Anx	Self	Infreq	Total T	Free T	Non-SHBG T	Prol	LH	FSH	Cort
Waist	1.00	+0.902 **				+0.462 *				+0.796 **			
Mean		1.00											
EAT			1.00										
Anx				1.00									
Self					1.00					-0.705 **			
Infreq						1.00							
Total T							1.00	+0.9206 **	+0.909 **		-0.480 *		
Free T								1.00	+0.971 **		-0.517 *		
Non-SHBG T									1.00		-0.468 *		
Prol										1.00			
LH											1.00		
FSH												1.00	
Cort													1.00

* Denotes significance, $p < 0.05$

** Denotes significance, $p < 0.01$

APPENDIX 9

SPEARMAN RANK ORDER CORRELATIONS PONDURAL INDEX WITH MEASURED HORMONE, PERSONALITY AND BODY IMAGE ESTIMATION

Group	Waist	Mean	EAT	Anx	Self	Infreq	Total T	Free T	Non SHBG T	Prol	FSH	LH	Cortisol
n = 26 40+ mpw	+0.203	+0.166	+0.266	+0.224	-0.148	+0.07	-0.217	+0.320*	+0.295	-0.316	-0.078	-0.179	+0.191
n = 18 20-40 mpw	-0.216	-0.318	-0.773	+0.304	-0.411*	+0.057	-0.397*	-0.360	-0.310*	-0.131	-0.06	+0.332	+0.469*
n = 11 Controls	-0.057	-0.120	+0.205	+0.264	-0.29	+0.457	-0.724*	-0.572*	-0.270	-0.508	+0.31	+0.2	+0.225

*Denotes significance, $p < 0.05$

SPEARMAN RANK ORDER CORRELATION OF NUMBER OF RUNS PER WEEK BY GROUP WITH BODY IMAGE, PERSONALITY AND HORMONE MEASURES

Group	Waist	Mean	EAT	Anx	Self	Infreq	Total T	Free T	Non SHBG T	Prol	FSH	LH	Cortisol
n = 28 40+ mpw	+0.023	+0.054	+0.549**	+0.016	-0.060	+0.247	-0.199	+0.155	+0.176	-0.149	-0.108	-0.108	+0.167
n = 21 20-40 mpw	+0.043	+0.082	+0.152	+0.025	-0.188	-0.048	-0.151	-0.210	-0.265	-0.222	-0.235	+0.125	+0.054

**Denotes significance, $p < 0.01$

mpw = miles per week

APPENDIX 10

RADIOIMMUNOASSAY FOR SERUM TESTOSTERONE

Reagents

1. Dilute buffer: 0.005% rabbit gamma globulins in 0.1 m,phosphosaline-gelatin buffer pH 70. Stability: 8 weeks at 4-8°C.

2. Anti-testosterone: testosterone-19-carboxymethylether-BSA was used to generate antiserum in rabbits. Antiserum binds 50-60% of testosterone ^{125}I derivative in the absence of non-radioactive testosterone. Antiserum was diluted with diluent buffer (A). Stability: 8 weeks at 4-8°C.

3. Testosterone standards: 0.0, 0.1, 0.25, 0.5., 1.0, 2.5, 5, 10 ng/ml diluted with testosterone free serum. Stability: 4 weeks at 4-8°C or 10 weeks at -15°C.

4. Precipitating antiserum (second antibody) goat anti-rabbit gamma globulins in 0.01 m phosphosaline buffer pH 7.5. 0.1 ml is sufficient to precipitate all the antibody bound antigen at room temperature in a minimum of 60 mins. Stability: 8 weeks -15°C.

5. Testosterone binding globulin inhibitor solution (TuBl solution). Stability: 8 weeks at 4-8°C.

6. Testosterone ^{125}I derivative contains less than 3 uCi per vial for a 100 tube kit. Diluted with diluent buffer (A). Stability: 8 weeks at 4-8°C.

Procedure

1. Set up tubes in duplicate.

2. Bring reagents to room temperature prior to use.

3. Add 0.5 ml of diluent buffer to non specific binding tubes.

4. Add 50 ul of 0.0 ng/ml standard to tubes 1, 2, 3, 4.

5. Add 50 ul (in duplicate) of each testosterone standard (0.1 ng/ml - 10 ng/ml) to tubes 5-18.

6. Add 50 ul (in duplicate) of control serum, female serum or diluted male serum to tubes 19 to end of assay.

7. Add 0.1 ml TGBl solution to all assay tubes and mix by shaking test tube rack for 10 seconds.

8. Add 0.5 ml of testosterone ^{125}I to all tubes.

9. With the exception of the non specific binding tubes, add 0.5 ml of anti-testosterone to all the tubes.

10. Vortex mix and incubate at 37°C for 120 minutes.

11. After 120 minutes incubation, add 0.1 ml of second antibody to all the tubes. Vortex, mix and incubate at 37°C for 60 minutes.

12. Centrifuge all tubes at 2300-2500 rpm for 15 minutes. Aspirate/decant supernatant.

13. Count precipitate in a gamma counter.

Standard Curve

A standard curve is set up using % bound standard to the first antibody.

Calculation of Percent Binding of Samples to Antibody

$$\%{}^{B}/_{B_o} = \frac{\overline{\text{CPM}}\text{ (sample) } - \overline{\text{CPM}}\text{ (blank NSB)}}{\overline{\text{CPM}}\text{ (0 standard } - \overline{\text{CPM}}\text{ (blank (NSB)}}$$

$\overline{\text{CPM}}$ = Average counts of duplicate

Sample = Particular serum or standard being calculated

Blank (NSB) = Blank tube (non specific binding tube)

0 Standard = 0 tube (also known as 100% binding tube)

Plot percent bound using 100% as the starting point against the testosterone standards, 0.1 - 10 ng/ml. Samples are converted as above and ng/ml sample concentration read directly off the curve.

Performance Characteristics

(i) Specificity:

Steroids	% Cross Reaction
Testosterone	100.00
5 α DHT	10.30
11, Oxotestosterone	2.00
5 α Androstane 3B, 17 B diol	2.20
(others minimals, cross reactivity)	

(ii) Precision/Reproducibility:

within assay variability 4-2 - 9.0%
between assay variability male - 7.5%
female - 12.6%

(iii) Accuracy: recovery mean = 96.8 - 98.6%

(iv) Sensitivity: 0.2 ng/ml

RADIOIMMUNOASSAY FOR CORTISOL

Reagents

1. 50 or 100 rabbit anti-cortisol coated tubes. Storage: 2-4°C. Stability: 90 days.
2. 55 or 110 ml of PBS/cortisol ^{125}I solution: 0.01 m phosphate, 0.85% sodium chloride pH 7.5 with 0.05% sodium azide (preservative). Contains 2.5/5 uCi of cortisol ^{125}I. Store: 2-4°C. Stability: 60 days.
3. Cortisol standards: 0.0, 1.0, 3.0, 10.0, 30.0, 100.00 ug/dl. Storage: 2-4°C or frozen. Stability: 90 days frozen.

Assay Procedure

1. Decant storage buffer from antibody coated tubes.
2. Pipet 25 ul of standard, sample or control, in duplicate.
3. Add 1.0 of cortisol ^{125}I/PBS solution.
4. Vortex all tubes briefly.
5. Incubate all tubes at 37°C for a minimum of 45 minutes.
6. Aspirate or decant tubes in same order as prepared.
7. Count tubes for one minute.

Calculations

A standard curve is set up %B/B_0 v concentration of standards. Samples are converted to a %B/B_0 ratio and read directly from the curve.

Compute %B/B_0 as follows:

$$\%B/B_0 = \frac{\text{CPM unknown}}{\text{CPM of 0.0 ng/dl standard x 100}}$$

Performance Characteristics

(i) Specificity:

Hormone	% Cross Reaction
Cortisol	100.00
Prednisolone	34.80
11-desoxycortisol	13.5
Prednisone	2.1
Cortisone	2.0
Corticosterone	10.7

(ii) Precision/Reproducibility:

interassay variation r = +0.992 - 0.986

within assay variation 7.9 - 8.8%

(iii) Accuracy: mean recovery 109%

(iv) Sensitivity: 1 ug/dl

RADIOIMMUNOASSAY FOR PROLACTIN

Reagents

1. Prolactin antiserum - contains lyophilized human prolactin antiserum (rabbit), 0.01 m phosphate buffer, rabbit gamma globulin, BSA, EDTA, inert dye and less than 0.1% sodium azide.

2. Prolactin ^{125}I - contains approximately 2 uCi lyophilized human prolactin ^{125}I, 0.01 m phosphate buffer, BSA, EDTA, inert dye, and less than 0.1% sodium azide.

3. Prolactin standard 0 ng/ml - contains 0.01 m phosphate buffer.

4. Prolactin standards - 2, 5, 10, 20, 50, 150 ng/ml. Contains lyophilized phosphate buffer, human protein, BSA, EDTA, less than 0.1% sodium azide and 2.0, 5.2, 10.0, 20.0, 50.0 and 150 ng/dl human prolactin.

5. Anti-rabbit gamma globulin - contains lyophilized anti-rabbit gamma globulin (sheep) bovine serum and gamma globulin, 0.01 m phosphate beffer, inert dye, and less than 0.1% sodium azide.

6. PEG solution - contains 130 ml 8% polyethylene glycole (MW 6,000) in phosphate buffer, less than 0.1% sodium azide.

7. RIA buffer pH 7.5 - contains lyophilized 0.01 m phosphate buffer, BSA, EDTA and less than 0.1% sodium azide.

8. Serotest quality control serum - contains lyophilized normal human serum.

Procedure (3 hour assay)

1. Pipet 0.1 ml prolactin standards, patient samples, and control serum appropriately labeled tubes.
2. Pipet 0.2 ml 0 ng/ml prolactin standard into NSB tubes.
3. Add 0.1 ml prolactin ^{125}I to all tubes.
4. Add 0.1 ml prolactin anti-serum to all tubes except NSB and total count tubes.
5. Vortex all tubes and incubate for 3 hours ± 5 minutes at room temperature (15° to 24°C).
6. Pipet 0.1 ml anti-rabbit gamma globulin into all tubes except total count tube.
7. Add 1.0 ml cold PEG solution to all tubes except TCT. Mix thoroughly by vortexing.
8. Centrifuge all tubes at room temperature, except TCT for 15 minutes at 1500 to 3000 xg.
9. Decant supernatant from all tubes except TCT. Blot rim of each tube to remove excess liquid.
10. Count each tube for 1 to 2 minutes in gamma scintillation counter.

Calculations

(a)
$$\%\ \text{NSM} = \frac{\text{CPM of NSB}}{\text{CPM of TCT}} \times 100$$

(b)
$$\%\ \text{Bound} = \frac{\text{CPM of 0 standard - CPM of NSB}}{\text{CPM of TCT}} \times 100$$

(c) CPM is corrected in the above calculations by

Corrected CPM = Average CPM - Average CPM of NSB

(d)
$$\%{}^{B}/_{B_0} = \frac{\text{Corrected CPM}}{\text{Corrected CPM of 0 ng/ml standard}} \times 100$$

(e) Standard curve is plotted by plotting $\%B/B_0$ on linear scale v concentration of the logarithmic scale.

(f) Sample concentration are read directly off curve

Performance Characteristics

(i) Specificity:

Hormone	% Cross Reaction
hPRL	100
hCG	< 0.0075 (2nd IS-HCG)
hFSH	< 0.03
hGH	0.13
hTSH	< 0.06

(ii) Precision/Reproducibility:

within variability 3.4 - 4.0 Method 1
2.3 - 5.3 Method 2

between assay variability 6.2 - 13.3 Method 1
6.2 - 15.2 Method 2

(iii) Accuracy: Method 1 91.6 - 97.2%
Based on Recovery Method 2 97.3 - 113%
} varies according to prolactin added, 5-150 ng/ml

(iv) Sensitivity: Method 1 (3 hr) 1 - 2 ng/ml
Method 2 (overnight) 0.5 - 1 ng/ml

RADIOIMMUNOASSAY FOR LUTEINIZING HORMONE

Reagents

1. LH I 125 - not more than 3 uCi I 125 and 30 mg bovine serum albumin in 5.5 ml solution.

2. LH Antiserum (rabbit) - approximately 1 ul normal rabbit serum and 60 mg bovine serum albumin in 11 ml solution, anti-serum sufficient to bind at least 15% of 40 ng LH.

3. Second Antibody Reagent (Amerlex) (donkey) - anti-rabbit antibody coated on polymer particles of uniform diameter sufficient to bind at least 22 ug rabbit γ-globulin in approximately 110 ml solutions, 3.0 ml normal human serum, EDTA.

4. Standard LH - 3.7, 9.2, 30, 92, 310 mIU/ml 2nd IRP-HMG and 50 mg bovine serum albumin in 1 ml solution.

5. Zero Standard - 250 mg bovine serum albumin in 5 ml solution.

Procedure

1. 200 ul zero standard were pipetted into Non-Specific-Binding tubes.
2. 100 ul of the standards were pipetted into Standard tubes.
3. 100 ul of the samples were pipetted into Sample tubes.
4. 100 ul of LH anti-serum were pipetted into all Standard and Sample tubes.
5. Tubes were vortexed, covered with plastic film, and incubated in a waterbath at body temperature for 1 hour.
6. 100 ul LH I 125 was added to each tube above and to Total Counts tubes.
7. Tubes were vortexed, covered with plastic film and incubated in a waterbath at room temperature for 1 hour.
8. 1.0 ml of second antibody reagent was added to all but the Total Counts tubes.
9. Tubes were vortexed and left for 10 minutes at room temperature.
10. Tubes were centrifuged at 1500 g for 15 minutes.
11. Supernatant was drawn off using suction.
12. Tubes were counted in a gamma counter for 2 minutes.

LH Assay Performance Characteristics

(i) Specificity: determined by cross-reactivity with the highly purified structually related human glycoprotein hormones.

Hormone - % Cross-reactivity

LH - 100.0
FSH - 2.4
TSH - 3.8
HCG - 19.0

(ii) Precision Reproducibility:

within assay variability	3.5%
between assay variability	6.6%

(iii) Accuracy: recovery experiments resulted in a mean recovery of 104%

(iv) Sensitivity: the smallest amount of LH which could be distinguished from zero as defined by the 95% confidence limits of the within assay variation of the zero standard was 2.0 mIU/ml 2nd IRP-HMG.

RADIOIMMUNOASSAY FOR FOLLICLE-STIMULATING HORMONE

Reagents

1. FSH I 125 - not more than 3 uCi I 125 and 30 mg bivone serum albumin in 5.5 ml solution.
2. FSH Antiserum (rabbit) - approximately 1 ul normal rabbit serum and 60 mg bovine serum albumin in 11 ml solution, anti-serum sufficient to bind at least 15% of 40 ng FSH.
3. Second Antibody Reagent (Amerlex) (donkey) - anti-rabbit antibody coated on polymer particles of uniform diameter sufficient to bind at least 22 ug rabbit γ-globulin in approximately 110 ml solution, 3.0 ml normal human serum, EDTA.
4. Standard FSH - 1.5, 5.5, 19, 61, 320 mIU/ml 2nd IRP-HMG and 100 mg bovine serum albumin in 2 ml solution.
5. Zero Standard - 250 mg bovine serum albumin in 5 ml solution.

Procedure

1. 300 ul zero standard were pipetted into Non-Specific-Binding tubes.
2. 200 ul of the standards were pipetted into Standard tubes.
3. 200 ul of the samples were pipetted into Sample tubes.
4. 100 ul of FSH anti-serum were pipetted into all Standard and Sample tubes.
5. Tubes were vortexed, covered with plastic film, and incubated in a waterbath at body temperature for ½ hour.
6. 100 ul FSH I 125 was added to each tube above and to Total Counts tubes.
7. Tubes were vortexed, covered with plastic film and incubated in a waterbath at body temperature for 1 hour.
8. 1.0 ml of second antibody reagent was added to all but the Total Counts tubes.
9. Tubes were vortexed and left for 10 minutes at room temperature.
10. Tubes were centrifuged at 1500 g for 15 minutes.
11. Supernatant was drawn off using suction.
12. Tubes were counted in a gamma counter for 2 minutes.

FSH Assay Performance Characteristics

(i) Specificity: determined by cross-reactivity with the highly purified, structurally related human glycoprotein hormones.

Hormone - % Cross-reactivity

FSH - 100.00
TSH - 0.35
LH - 0.10
HCG - < 0.03

(ii) Precision-reproducibility:

within assay variability	4.8%
between assay variability	9.2%

(iii) Accuracy: recovery experiments resulted in a mean recovery of 104%.

(iv) Sensitivity: the smallest amount of FSH which could be distinguished from zero as defined by the 95% confidence limits of the within assay variation of the zero standard was 0.8 mIU/ml 2nd IRP-HMG.

ASSAY FOR SERUM SEX HORMONE BINDING BETA GLOBULIN BINDING CAPACITY (SHBG-BC)

Principle

Modification of method of Rudd et al. (1974), involving selective removal of endogenous steroids occupying SHBG binding sites, with a charcoal suspension, under defined optimum conditions (without affecting ^{14}C-T binding to SHBG). This is followed by a short incubation with a single dose of ^{14}C-T, which saturates all SHBG binding sites. Precipitation of the SHBG-bound tracer is accomplished by selectively separating it from albumin-bound and free tracer with a 50% saturated (final concentration) ammonium sulphate solution. The precipitated SHBG-bound ligand is counted.

Reagents

1. [4-^{14}C]-testosterone, specific activity 51.9 mCi/mmol (New England Nuclear). Dilute in redistilled methanol, such that 0.05 ml contains 2200 DPM (1 nCi). Store at 4°C.

2. Norit 'A' charcoal (Sigma Chemicals, St. Louis, MO), as a suspension (50 mg/ml) in deionized water. Transfer 5 gm Norit 'A' charcoal to 100 ml water and mix with an electromagnetic stirrer. Prepare fresh for each assay.

3. A 44% (w/v) ammonium sulphate solution - add 440 g reagent grade $(NH_4)_2\ SO_4$ crystals (Sigma) to 1000 ml deionized water. Store and use at 4°C.

4. Steroid assay buffer - heat 1000 ml deionized water to 50°C and add 2 g gelatin (275 Bloom Fisher Scientific). Allow dissolution, then add 17.4 g sodium phosphate dibasic, 10.8 g sodium phosphate monobasic, and 18 g sodium chloride, and swirl until in solution. Make to a total volume of 2000 ml, then add 2 g sodium azide when buffer has cooled to room temperature, and adjust pH to 7.0.

5. Liquid Scintillation Fluid - Readi Solv IV (Beckman, Fullerton, CA).

Assay

After mixing the charcoal solution for at least 20 minutes, transfer 0.4 ml of the suspension (while still mixing) to 12 x 75 mm tubes containing 0.4 ml of serum sample. Vortex, then stand the mixture at room temperature for 30 minutes. Centrifuge for 15 minutes at 3000 rpm. Take duplicate 0.2 ml aliquots of the supernatant without disturbing the charcoal button, and transfer to 12 x 75 mm tubes. Add 50 uL of ^{14}C-T in methanol solution to each tube, vortex, and incubate at 37°C for 2 hours. To 4 counting vials, add 50 uL tracer solution as standard, as well as 0.1 ml $(NH_4)SO_4$ solution. Put tubes on ice, wait 4 to 5 minutes, then add 4 volumes (1 ml) of cold ammonium sulphate solution, such that the final concentration of the mixture is 50% saturated. Parafilm, vortex, then centrifuge at no less than 3000 rpm for 20 minutes at 4°C. Aspirate the supernatant with a Pasteur pipette attached to vacuum pump without disturbing the protein precipitate. Add 0.2 ml of steroid assay buffer to the precipitate, vortex, then transfer 0.1 ml aliquots of this solution to scintillation vials. Add 5 ml of scintillation fluid to each vial, cap tightly and shake for 5 minutes, then count on a liquid scintillation counter for 5 minutes.

Calculations

Assuming the best estimate of the molecular weight of SHGB is 10^5 g/mol, and one binding site per molecule, normal serum contains 1.7 mg/L SHBG (17 nmol/L). Therefore, since 0.1 ml of serum is used, the normal SHBG content for 0.1 ml is approximately 1.9 pmol. Since 19-19.5 pmol of ^{14}C-T provides saturation, the molar ratio of T:SHBG at this dose is approximately 11:1. 19 to 19.5 pmol represents between 5.5 and 5.6 ng. At a specific activity of 51.9 mCi/mmol, the quantity of [4-^{14}C] - testosterone that must be added to 0.1 ml of charcoal treated serum is 2200 dpm. To ensure saturation of pregnancy sera requires 95 pmol (27.4 ng), equivalent to 10870 dpm.

$$\frac{E}{C} \times N \times \frac{100}{V} \times \frac{1}{1000} \begin{array}{l} = \text{ug/100 ml serum} \times .0347 \\ = \times 10^{-8} \text{ mol l}^{-1} \text{ T bound,} \end{array}$$

where E = CPM in aliquot of bound fraction counted
C = standard CPM in 50 uL tracer solution
N = ng of ^{14}C-T added in 50 uL tracer solution
V = final volume of serum in the assay = 50 uL

CALCULATION OF FREE TESTOSTERONE AND NON-SHBG-BOUND TESTOSTERONE

Non-SHBG-bound testosterone and free testosterone were calculated using an equation based upon the law of mass action and assuming two binding systems. Testosterone-SHBG and testosterone-albumin association constants were taken from the literature (Dunn et al., 1981).

Association Constants

(1) T - SHBG 16×10^{8} l mol^{-1}
(2) T - Albumin 4×10^{4} l mol^{-1}

Assumed albumin concentration 5.6×10^{-4} l mol^{-1}

Equations

(a) Distribution of bound testosterone between two competing proteins P_1 (SHBG) and P_2 (albumin) were described by the following:

$$\frac{S6_1}{S6_2} = K_1 \frac{(n_1P_1 - S)}{n_2K_2P_2}$$

where:

S = total steroid
K_1, K_2 = association constants
P_1 = SHBG
P_2 = albumin

From this was gained the ratio of SHBG to albumin bound testosterone.

Total bound testosterone was taken as the sum of the ratios. Percentage of total testosterone bound to proteins can thus be derived.

(b) Free testosterone was calculated via

$$\frac{Sb}{Su} = K_1 (P_1 - S_1) + K_2P_2$$

$$\therefore \quad Su = S\left[\frac{1}{1 + \left(\frac{Sb}{Su}\right)}\right]$$

where:

Sb = total bound

Su = free testosterone

K_1, K_2 = association constants

P_1, P_2 = binding proteins